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THE CLINICAL ASPECTS OF BRANCHIAL FISTULÆ.

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IN a fœtus approximately thirty days old at least four well-developed grooves can be seen on the side of the neck. Commonly these are termed 'branchial clefts' (*Fig.* 105), and the intervening bars 'branchial arches'. In human embryos these clefts are, correctly speaking, grooves — grooves on the outside, and on the pharyngeal aspect separated by a layer of mesoblast. The clefts begin to appear about the tenth day, when the embryo is about 2·6 mm. in length. Forty days later, or even in less time, the second, third, and fourth clefts have disappeared. There can be little wonder that in studying so transitory and minute a structure as the branchial apparatus in material which is difficult to obtain, differences of opinion have arisen concerning the origin of branchial fistulæ.

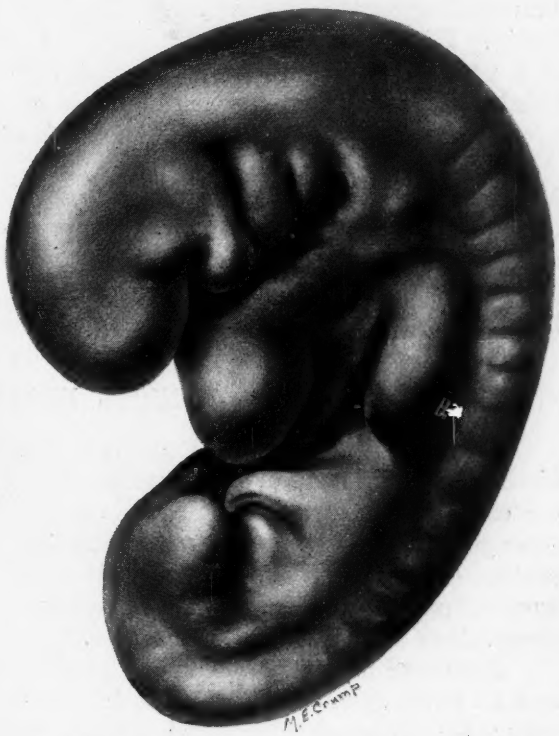


FIG. 105.—Fœtus showing branchial grooves and arches.

ORIGIN OF BRANCHIAL FISTULÆ.

Rabl's Theory. — The one basic fact to which all embryologists subscribe is that the Eustachian tube is the persistent first cleft, and that the tympanic membrane represents the septum between the external and the internal grooves. Rabl[1] homologized a complete branchial fistula with the auditory canal, suggesting that the septum (tympanic membrane) had perforated early

in embryonic life. From a study of anatomical relationships Rabl agreed with other observers that the cleft usually implicated was the second. This has been termed the 'arm-chair' theory. Until it has been definitely disproved its simplicity is bound to make an appeal to the clinical worker. It is a seductive theory; one merely has to assume that the branchial grooves are the atavistic remnants of the fish's gills, and lo! the patient with a branchial fistula before us becomes one more example illustrating Crile's[2] conception of man as a giant amœba slowly creeping up the slippery sides of time.

Many of those who are best able to judge[3–5] deride this theory. J. E. Frazer,[3] whose works on embryology are so well known, objects to the very word 'branchial'. He states that branchial fistulæ have nothing to do with the branchiæ, and in many cases are outside the region which might be homologized with the gills of lower vertebrates.

Wenglowski's Theory.—This is the very antithesis of an 'arm-chair' theory. This patient Russian[4] worked for five years on the branchial apparatus, examining no fewer than 75 embryos and 246 cadavers before propounding his theory in 1912. An opinion based upon premises such as these justly commands respect. He presented evidence to show that the branchial apparatus cannot leave remnants in the neck below the level of the hyoid. His conclusion is that a branchial fistula, if the term may be allowed, is a persistent thymic duct, and that this thymic duct originates from the third pharyngeal pouch, whence it pursues a long oblique course down the neck, to reach the upper part of the back of the sternum.

Even if I were qualified to do so, it is not my desire to become involved in the intricacies of the embryology of this difficult region. My object has been to approach the subject from the purely clinical side, but I have fostered a hope that by dropping a morsel of truth from the patient's neck into the seething cauldron of controversial cervical embryology, perchance I might help to clarify what is to be served to the student to-morrow.

CLINICAL FEATURES.

Unlike a thyroglossal[7] fistula, a branchial fistula is usually present from birth. While in none of my cases has there been any hereditary factor, there are cases on record where the abnormality has been transmitted,[8–10] usually through the mother. Females appear to be more often affected in a proportion of 3 : 1. When the fistula is unilateral the right side is said to be more often affected. It happens that in the five unilateral cases under my observation all have been right-sided.

Branchial fistulæ are of three distinct types: (1) Complete, with an outer and an inner opening; (2) Incomplete external fistula, with an outer opening only; (3) Incomplete internal fistula, with an inner opening only. The commonest variety is blind internally, the rarest blind externally.

The External Opening.—In fully 80 per cent of those cases which have an external orifice that orifice is situated in the lower third of the neck, opposite the anterior border of the sternomastoid (*Figs.* 106, 112). In this connection it is interesting to compare and contrast a typical branchial cyst[11, 12] with a typical branchial fistula.

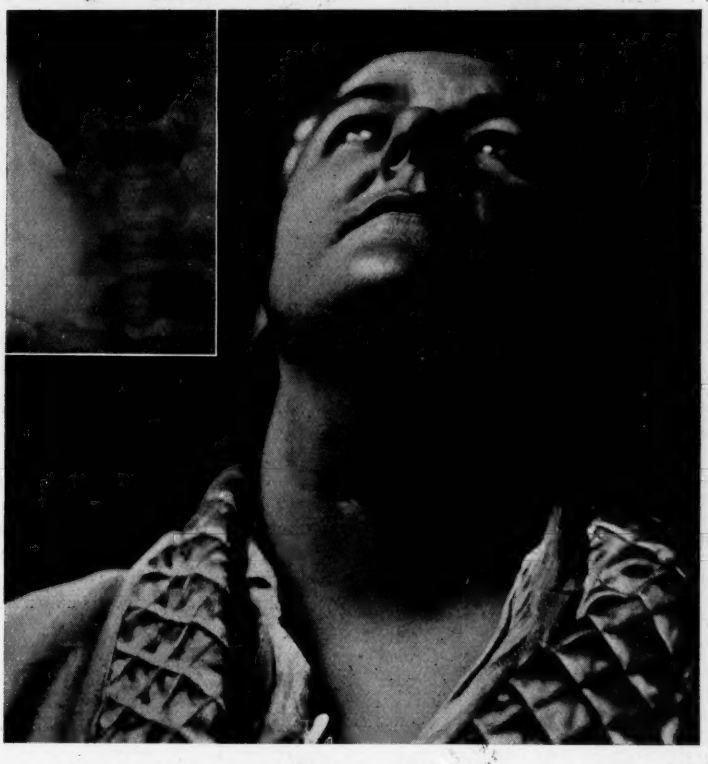


FIG. 106.—A branchial fistula of over thirty years' duration. *Inset*: X-ray after injecting the fistula with lipiodol.

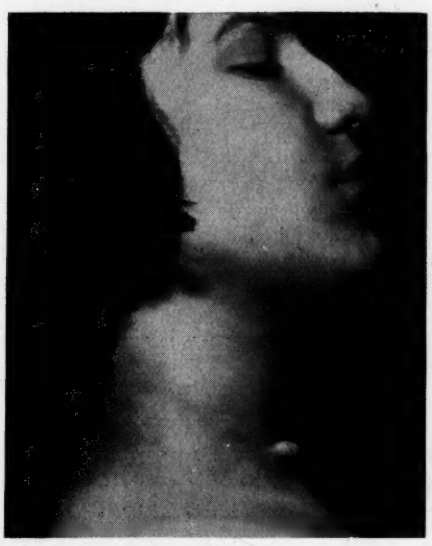
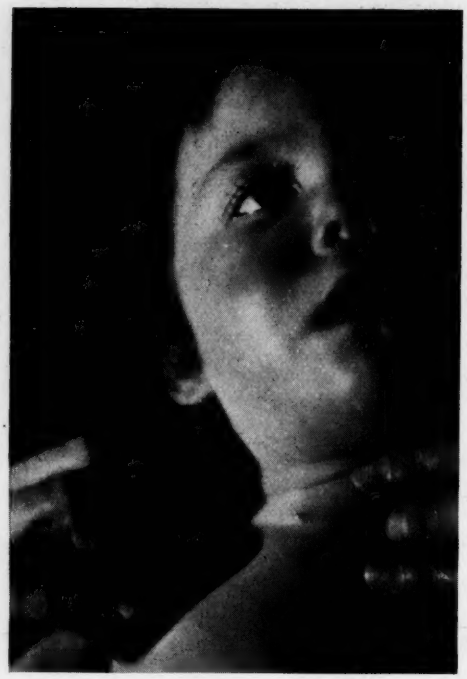


FIG. 107.—Cervical auricle. These appendages are believed to have been common in the days of the Romans, for Sir John Bland-Sutton has drawn attention to examples of statuary of this period where large bilateral cervical auricles are depicted by the sculptor—proof, indeed, that they were common enough to attract notice.

Fistula	Cyst
1. Present at birth.	Appears in early adult life.
2. Situated in the lower third of the neck.	Situated in the upper third of the neck.
3. Lined with columnar epithelium, usually ciliated.	Lined with stratified squamous epithelium.
4. Exudes sticky mucus.	Filled with opaque fluid rich in cholesterol.



The lower third of the neck opposite the anterior border of the sternomastoid is also the situation *par excellence* of two other rare branchial abnormalities—cervical auricle (*Fig.* 107) and persistent branchial cartilage[13]

Fig. 108.—A persistent branchial cartilage. It lay in the subcutaneous tissue tethered to a dimple in the skin situated where a branchial fistula commonly opens. By stretching the skin, as was done while this photograph was taken, the cartilage became more apparent.

(*Figs.* 108, 109). These structures can be homologized clearly with the pinna.

Atypical External Openings.—In a small proportion of cases the external orifice is situated midway along the anterior border of the sternomastoid. Very occasionally the orifice has been observed at the posterior border of that muscle; in a figure by Semken[14] the orifice is shown in the posterior triangle of the neck. In assessing atypical external apertures we must inquire into the possibility of the fistula being acquired, an example of which is afforded by the following case :—

Fig. 109.—Section of the branchial cartilage in *Fig.* 108.

Mrs. G. C., aged 35, was sent complaining of a fistula which discharged mucus and pus (*Fig.* 110). Twenty years previously a swelling in the neck had been incised, and the resulting sinus continued to discharge. At varying intervals the discharge ceased, and this was usually followed by subacute inflammation on the right side of the neck. The indurated fistula, which extended up to the base of the skull, was dissected out in the manner presently to be described. The result was entirely satisfactory.

FIG. 110.—An acquired branchial fistula of over twenty years' duration. A branchial cyst had been incised during one of the periodic attacks of inflammation to which these cysts are subject.

An Internal Orifice.—For a fistula to have an internal orifice is distinctly uncommon. Such an opening is usually situated in the region of the tonsil,[15] most often in the fossa of Rosenmüller (*Fig.* 111). The relation of the tonsil to the innermost limit of a patent or potential branchial fistula is important. Kramer[16] reported a case of an internal cervical fistula which followed tonsillectomy. I have seen a branchial cyst appearing for the first time a fortnight after tonsillectomy, and Johnson[17] reported a similar case.

FIG. 111.—Complete branchial fistula extending from the lower third of the neck to the posterior tonsillar recess.

SYMPTOMS.

Apart from the continual or intermittent discharge of mucus, which is somewhat annoying, the leading symptoms are due to recurrent attacks of inflammation in the fistula. In cases with an external orifice these inflammatory attacks are fairly constant, and often it is during one of these attacks that the patient seeks advice (*Fig.* 112).

The orifice of the fistula is commonly small and relatively inconspicuous, although a few cases have been described when a complete fistula was so large

FIG. 112.—Bilateral branchial fistulæ; left side inflamed.

that the patient's complaint was that bread crumbs and other particles of food passed through it.[18]

Sometimes the vagus is a direct relation of the fistula, and probing, pinching, or injecting these fistulæ has produced irritative vagal symptoms such as cough, palpitation, and an intermittent pulse. Carp[19] described the case of a boy of 5 with a branchial fistula who gave a history of an unproductive cough of two years' duration unsuccessfully treated by tonsillectomy and medical measures. At operation the tract was found adherent to the vagus, and excision of the fistula cured the cough. Similar examples have been reported.

The externally blind variety appears to be rare. It is possible that they

are more common than is generally supposed, and that a fine-calibred tract discharges mucus unobtrusively into the fossa of Rosenmüller. Douglas[20] reports a case of a man who, when he closed his mouth and occluded his nostrils, could force air into his blind fistula and distend his neck. When this variety of fistula is complicated by having a large lumen it is liable to become a source of danger. Eddowes's patient was a nurse of 30 whose fistula became periodically full of purulent material, and absorption of toxins from the pus sac undermined the patient's health.

INVESTIGATION AND TREATMENT.

If the tract shows evidence of recent inflammation it should be treated conservatively for at least fourteen days.

To ascertain the extent of the fistula there is no better method than a lipiodol injection. The following method of investigation and treatment has several notable advantages. Three or four hours before the proposed operation for complete extirpation of the fistula the skin about the orifice is infiltrated with local anæsthesia, and a purse-string is introduced *very close to the periphery of the opening*. At this stage the suture is not tied. If the external orifice will admit a ureteric catheter, this is introduced. More often it will be found that the opening is too small, when a lachrymal-duct syringe can be employed (*Fig.* 113). Lipiodol does not run freely through a fine-nozzled syringe, but diluted with an equal volume of liquid paraffin and well stirred it will be found to flow tolerably, while the diluted medium's opacity to X rays is in this instance sufficient to cast a good shadow. During the injection due regard must be given to possible reflex vagal symptoms; I have perhaps been fortunate not to have observed any disturbances from this cause. When it is judged that the sinus is distended with the medium the syringe is removed while the purse-string suture is drawn taut and tied. The patient can now be sent to the radiological department and X-rayed without the annoyance of the lipiodol escaping, which only too often occurred when employing other techniques. The pictures obtained by the method are instructive (*see Fig.* 106, *Inset*).

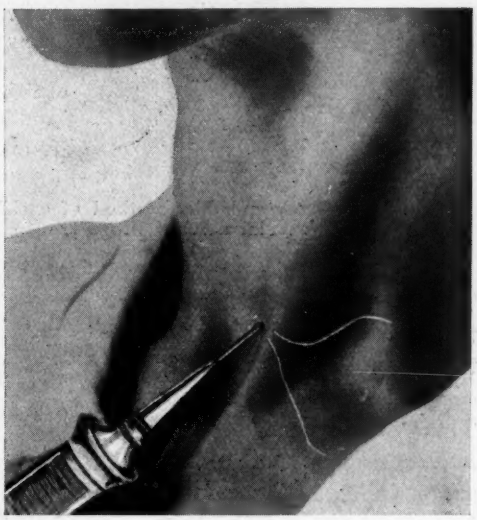


FIG. 113.—Method of injecting a cervical fistula with lipiodol. A subcutaneous purse-string suture has been inserted and a lachrymal-duct syringe is being employed.

Complete Extirpation of a Branchial Fistula.—The neck and the purse-string stitch have been kept sterile during the time (usually a few hours) which intervenes between the X-ray and the operation. An oblique, mainly transverse, incision is made about the external orifice, taking care that the

purse-string suture is not cut in the process (*Fig.* 114). Once the skin is freed about the orifice of the fistula, the free ends of the purse-string can be caught in a hæmostat, and in this manner gentle traction can be applied to the fistula. Instead of the tract becoming more and more difficult to dissect, it will be found usually that sinuses distended with lipiodol, which converts them into elongated cysts, can be freed from surrounding structures with comparative ease. So the dissection proceeds until mild traction upon the fistula and reasonable retraction of the upper limit of the wound fails to give the most perfect exposure.

The time has now come when a second incision should be made in the skin parallel with the first, but at a higher level (*Fig.* 115).

FIG. 114.—The step-ladder method of excising a branchial fistula. Stage 1.

The tract is threaded through from the first to the second incision and the dissection is begun anew. This 'step-ladder' method leaves quite inconspicuous scars (*Fig.* 116), and robs the dissection of a branchial fistula of most of its terrors. The last part of the dissection is often rather difficult. I have noted on two occasions that the tract ran between the external and the internal carotid arteries to reach the pharyngeal wall. On another occasion the tract, which had been the seat of many attacks of inflammation, was intimately adherent to the jugular vein just below its bulb.

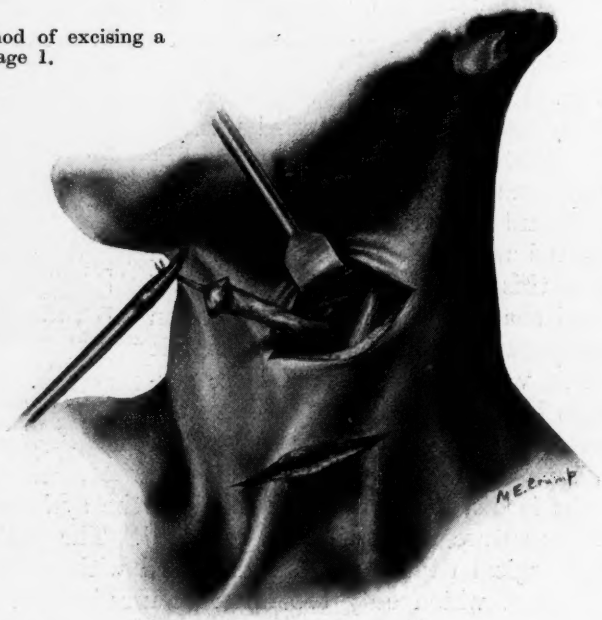


FIG. 115.—The step-ladder method of excising a branchial fistula. Stage 2.

Several operators have had to make a special dissection of the vagus from the wall of the fistula, which, if it is long enough, always appears to dive towards the pharynx beneath the posterior belly of the digastric. It behoves us to have adequate exposure, particularly during the last stages of the dissection, and the dual incision gives us just that exposure we need without leaving the patient, often a young woman, with a deforming scar.

FIG. 116.—Patient six weeks after excision of a branchial fistula by the step-ladder method.

The dissection completed, there remains only to close the skin according to individual preference. It is usually advisable to provide drainage through a stab-puncture wound below the first incision—that is, just above the clavicle. By employing the dual incision the less satisfactory and probably more dangerous methods of inverting the pharyngeal end[5, 21] of the fistula become unnecessary and need not be described. With proper exposure every fistula can be cleanly dissected from the neck. In bilateral cases it is distinctly unwise to attempt anything more than the extirpation of one fistula at a sitting.

FIG. 117.—Typical section of a branchial fistula, showing the ciliated columnar epithelium.

Histology of the Excised Tract.—Typically a branchial fistula is lined by columnar ciliated epithelium (*Fig.* 117). Such a lining is practically a constant finding unless there have been many recurrent attacks of inflammation in the fistula, in which case the epithelial lining may be partially or totally denuded. In a number of cases muscle fibres are present in the walls of the tract. As in the case of branchial cysts, there is often an ensheathing layer of lymphadenoid tissue.

CONCLUSIONS ON THE DERIVATION OF BRANCHIAL FISTULÆ FROM A CLINICAL STUDY.

1. Much as we may admire Wenglowski's assiduous labour, his statement that the branchial apparatus never leaves remnants in the neck below the level of the hyoid—a statement which is the keynote of his hypothesis—must be incorrect. *Figs.* 108, 109 illustrate a persistent branchial cartilage—a remnant, the origin of which cannot be disputed—situated in the lower third of a child's neck in the exact position where a branchial fistula commonly opens.

2. A cervical auricle, by its structure clearly a homologue of the pinna, is situated at the point where a branchial fistula usually opens.

3. Histologically a branchial fistula is usually lined by columnar epithelium, and its walls sometimes contain muscle. There is no reason why a thymic bud should contain these elements.

4. The anatomical relationships of a branchial fistula as displayed during an operation for its extirpation are in keeping with those depicted in works on embryology as belonging to the cervical sinus and the second cleft which persists in the goat to open into the fossa of Rosenmüller.

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THE DIVERTICULA OF THE JEJUNO-ILEUM.

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"The Diverticula and blind bypaths in
Which sophistry and deceit are wont to tread."
HALES—*Golden Remains.*

In the Century and Oxford Dictionaries a diverticulum is described as a 'way-side shelter or lodging', with, from the context, the underlying meaning that they are houses of ill repute where trouble is apt to brew. In the large intestine they live up to their bad reputation, and as a temporary lodging for bowel contents can give rise to endless trouble. As a contrast the small intestine is interesting, for the condition can be present to an advanced degree and yet the patient be free from suggestive symptoms. Such probably is natural, when one considers the rapidity of the intestinal stream and fluidity of the contents in the small as compared with the large intestine. The difference in the anatomical features of diverticula in the two regions is probably also a factor (*Table I*).

Table I.—Comparison of False Diverticula in Large and Small Intestine.

Large Intestine	Small Intestine
Sex.—Male preponderance 4–1.	Male preponderance 3–1.
Age.—Average 60.	Average 60.
Causation.—Spastic colon.	Increased intra-intestinal pressure.
Definitely related to situation of artery.	Definitely related to situation of artery.
Symptoms.—Left iliac fossa: discomfort. Constipation, flatulence. May be tumour. May be symptomless.	Vague indigestion. Flatulence, borborygmi. Occult blood. May be symptomless.
Complications.—Perforation with local or general infection. Adhesions, kinking, obstruction, fistula formation.	Perforation, local abscess or general peritonitis. Adhesions, kinking and obstruction.
Region.—Majority sigmoid colon, 70 per cent.	All start in upper jejunum.
Situation.—Between the longitudinal bands.	Mesenteric border.
Spread.—Into epiploic bodies and leaves of mesentery.	Into leaves of mesentery.
Size.—Cherry stone to cherry.	Cherry to orange.
Shape.—Retort-shaped.	Spherical or mushroom.
Neck.—Narrow in proportion—closed—firm margin.	Large in proportion—open.
Contents.—Inspissated fæces or mucin.	Fluids, gas, or empty.
Induration.—Usually present.	Nil.
X Ray.—Usually positive and definite.	Often negative. When positive conclusive.
Malignancy.—Possible forerunner.	No connection.
Diverticulitis.—Common.	Rare.
Perforation.—Common.	Rare.

It is not here intended to deal with all the diverticula found in the small intestine, but rather with one type only—the multiple false variety.

CLASSIFICATION OF JEJUNO-ILEAL DIVERTICULA.

Of the diverticula occurring in the jejuno-ileum there is no satisfactory classification. The writer would tentatively suggest the following as a basis from which to work, and one which, with greater knowledge, might be further subdivided: (1) Multiple false diverticula; (2) Meckel's diverticula; (3) Congenital enterogenous diverticula; (4) Anomalous diverticula.

Anomalous Diverticula.—Taking these in the reverse order, one would include under the last group those irregular sacs which occur along the intestine due to (*a*) the drag of a tumour, (*b*) the pull of adhesions, (*c*) the softening resulting from caseous glands or tuberculomata. Naturally such pouches may vary in size, shape, position, and appearance, depending upon the causative force. No clinical syndrome can be drawn up for them. They may not necessarily recur in individuals true to type, and are best included under the general term 'anomalous'.

Enterogenous Diverticula.—This group of diverticula has been dealt with in this JOURNAL very fully in an excellent article by Arthur Evans. Suffice it to say that these sacs arise primarily as isolated masses of cells, which separate off as small islands from the primitive gut. These islands by becoming vacuolated in the centre form cysts, and if later a secondary attachment is made to the intestine, they produce diverticula. Their interest lies in the fact that they are composed of a wall similar to the gut, i.e., muscle, mucous membrane, goblet cells, etc. They can occur at almost any point along the course of the small intestine attached to the mesenteric or antimesenteric border. Their origin helps to explain most of those isolated unique diverticula reported in the literature from time to time. In shape and appearance they may vary a great deal, yet a common etiology and pathology groups them together and allows them all to be included in the same subdivision.

Meckel's Diverticula.—It is said that from a survey of the vast amount of literature and the enormous number of monographs upon the subject the unobliterated vitello-intestinal canal has been found in man in about twenty-four different forms and stages of obliteration. If one realizes that each form discovered can have some ten to twenty possible sequelæ and complications, such as inflammation, gangrene, fistula, peritonitis, new growth formation, intestinal obstruction, tuberculous disease, etc., then one has only, by the simple mathematical procedure of permutation and combination, to see how many clinical forms Meckel's diverticula can assume to harass the surgeon. But as the disease has a constant origin irrespective of the sequela, it is put in a class by itself and will not be referred to further in this article.

Multiple False Diverticula.—The first group is the most interesting, as a common pathology exists for the disease in both small and large intestine. In the small gut the condition is rare; on going through 5000 case records in the Royal Victoria Hospital, Belfast, only one other case was discovered in addition to that operated upon by the writer, and the personal case is the

only one on record where not only the pre-operative but also the post-operative radiograms showed the condition. This was possible since all the diverticula had *not* been removed.

The case described is not an isolated example of a very rare disease, but rather a typical example of a disease well recognized but as yet receiving scant recognition in English text-books or by English writers in general. It is interesting to note that for diverticulosis of the large intestine the reverse holds good. It would appear to be a disease *par excellence* of the English-speaking people; certainly the best monographs on this subject are in that language.

Genuine cases of multiple diverticula of the small gut collected from the literature, when grouped together, form such a small collection that one is forced to realize that the condition is a rare one. Godard, who investigated the subject in France, believes that it is particularly rare in his country, and Moreau and Murdock writing upon the subject said, "Result of a post-mortem or surgical surprise—in all cases a disease deprived of all clinical interest—such for a long time was the total of our knowledge of small intestine diverticula."

Although the first case to be recorded in detail was in 1844 by an Englishman, Sir Astley Cooper, it is a noticeable fact that, excepting reports of individual cases such as those of Braithwaite and others, little work has been done upon the subject by Englishmen. In America J. T. Case has done some excellent work, especially from a radiographical standpoint, while in Germany Helvestine and in France Henri Godard have investigated the condition in considerable detail. It is an interesting fact that although Cooper described his case in 1844 in the post-mortem of a man who had died of cirrhosis of the liver, Chomel in 1710 had mentioned the first duodenal diverticulum, while diverticulitis of the colon, a much commoner disease, was not reported till 1849, and then by Cruveilhier.

The early cases were all found at post-mortem, but in 1914 Gordinier and Sampson discovered the first case in the living. The patient, a woman of 45, was suffering from intestinal obstruction due to kinking of the intestine at the site of a suppurating diverticulum. Until 1920 not a single case had been diagnosed by X rays, and in that year J. T. Case published two authentic examples. It is a noticeable fact that since then hardly six other cases have been so diagnosed notwithstanding the great improvement in X-ray technique.

Rarity.—The condition is a rare one. Until 1923 Sheppe had collected 30 cases from the literature of eighty years. Of the 26 collected by Watson, 18 were from post-mortems and 8 from operations. Godard collected 50 cases in all, and Case claims to have found 70, but some of these would be placed by other writers in one of the other subdivisions of jejuno-ileal diverticula.

Frequency.—The real frequency will never be exactly known, as so many cases exist unsuspected owing to an entire absence of clinical symptoms.

The small gut is often spoken of as the 'silent area of the intestinal canal', and when one sees upwards of five feet of jejunum surmounted on the mesenteric border by diverticula each as large as a hen's egg yet without symptoms, one realizes how true it really is.

Adams, writing in 1926 on the surgery of the small intestine, mentioned that he had never seen a single case.

Relative Frequency.—The relative frequency of diverticula of one part of the alimentary canal as compared with another is variously stated by different writers. The anatomist, pathologist, surgeon, and radiologist all give figures which differ considerably, as might readily be expected. Radiology is especially disappointing, as will be shown later.

Cases are on record, e.g., Stevenson's, where, although the gut was known to be covered with diverticula from the knowledge gained at a previous operation, yet repeated radiograms showed no diverticular shadows. Consequently it is easily understood that the radiologist's figures will underestimate the frequency of the condition. As regards the surgeon, his help is called in only in those cases diagnosed as such, or when the case, although unsuspected, is found during laparotomy. His figures will thus exclude all those—75 per cent of the total—who pass to their graves with the disease unsuspected. The anatomist's and pathologist's figures similarly will not give an accurate record.

From the above it is obvious how difficult it is to gather true statistics and how much one will vary from the other, although each in itself is a true and honest record. It will also help one to understand the variations in the figures which follow.

The frequency of diverticula of the alimentary tract is usually considered and is generally accepted as occurring in the following order of frequency: (1) Colon; (2) Meckel; (3) Duodenum; (4) Pharynx and œsophagus; (5) Stomach; (6) Jejunum. From this it will be seen that the disease under survey is the rarest.

Larimore and Graham's figures show that in 3446 complete intestinal opaque meal examinations diverticula were found in 105 cases: 9 œsophagus, 3 stomach, 19 duodenum, 3 jejunum, 71 colon.

J. T. Case in 6874 complete examinations found: 85 duodenum, 4 jejunal, 1 jejuno-ileal, 138 colon. And later in 1920, after 10,000 examinations, he found 10 cases in all in the small intestine.

Reinhardt, in 5000 post-mortems, found 3 cases only, ages 66, 84, and 86. In all there had not been any digestive symptoms. Statistics from Johns Hopkins Hospital, Baltimore, show that in 2600 autopsies there were found 13 cases of diverticula of the small intestine; these presumably included duodenal, but excluded Meckel.

Henri Godard reports the result of 13,069 post-mortems collected from the Dresden City Hospital, Bender Hygienic Laboratory, and Boston City Hospital. There were, apart from 39 cases of Meckel, 16 diverticula of the small intestine. His figures are not far distant from the 1 per 1000 as quoted by others.

Marxer, in a personal series of 1000 X-rays, had 140 diverticula in all: 38 duodenal, 6 jejunal, 7 ileal, 6 appendicular, 83 colon.

The discovery of these cases can be divided into three main periods:—

1. Those discovered at post-mortem 1844–1914; this was prior to the period of safe abdominal exploration. The condition was thought to be a mere curiosity, and as presumably the patients had had few, if any, symptoms

it was looked upon as a matter of little importance. Reinhardt's 3 cases, aged 68, 84, and 86, had had no digestive symptoms.

2. The second period, starting in 1914, was when the disease was discovered in the course of laparotomy for some other condition, e.g., gall-stones, pyloric stenosis, obstruction, etc. (Gordinier and Sampson, the writer, et al.)

3. The third period, starting in 1920, is the present, when, with perfection of radiography and its technique, it is hoped to make a firm preoperative diagnosis more often.

It will be thus seen that the disease should appeal to all branches of the profession, although the treatment of the acquired condition is preeminently surgical.

Moynihan, speaking of false diverticula, said, "the pouches may be numerous, rarely larger than a walnut, on the mesenteric side, and because of the large orifice do not as a rule retain food for long periods, and do not, therefore, often lead to serious complications."

Age Incidence.—As regards age, if one excludes one or two notable examples, the disease is one of old age, 70 and upwards. Case, in his series, found the average age for men was 66 and for women 60.

It is interesting to note that when complications arise they do so at an early age and are rarer in the older, so that whilst the uncomplicated cases were found chiefly post mortem and in the elderly, those cases with obstruction, inflammation, etc., appeared between the ages of 40 and 60.

The present case is exceptional in that the patient was 30 years of age.

Sex Incidence.—There is a preponderance of the male sex. In a series of 40 cases there were 29 males to 11 females. In fact, it is interesting to see in how many there was an enlargement of the prostate, leading in some to uræmia and death. The associated straining, with its resultant increase in abdominal pressure, was thought by many to be the *fons et origo mali*, but when one finds it in a female patient aged 30, as described here, one must look for other causes. In any disease found chiefly in men between the ages of 60 and 80 a co-existent hypertrophy of the prostate would exist, so that it is not safe to consider this necessarily a case of cause and effect.

Obesity was not mentioned in sufficient cases to allow of a definite statement. In the personal case recorded the patient outwardly was of average build, but her omental and mesenteric fat were very excessive. The leaves of the mesentery were separated by about $\frac{3}{4}$ in. of adipose tissue.

ANATOMY OF THE SMALL INTESTINE.

Prior to a study of the pathology of the condition, the anatomical features of the small intestine relevant to the present condition must be mentioned.

The small intestine is a long tortuous hollow muscular tube. Its average length is 22 ft. 6 in. in the male and in the female 6 in. longer. Extremes vary from a maximum of 31 ft. 10 in. to a minimum of 15 ft. 6 in. The outer wall is regular without sharp folds or sacculations. The diameter gradually decreases so that at the lower end it is only one-third of that at the beginning (Piersol). Four definite coats are easily distinguished—mucous (or lining), submucous, muscular, and serous (or covering).

The mucous coat contains glands, folds, and villi which aid its secreting and absorbing area. The epithelium covering the villi is a single layer of cylindrical cells which appear striated near the lumen. In many places over the villi mucus-producing goblet cells share the free surface. Lying between the ordinary epithelial elements migratory leucocytes can also be seen.

The stroma or tunica propria of the mucous coat resembles lymphoid tissue, being composed of a connective-tissue reticulum containing small round cells similar to lymphocytes. This stroma fills the space between the glands and forms the core of the villi over which the epithelium stretches. The deepest part of the mucous coat is occupied by a well-marked muscularis mucosæ in which an inner circular and outer longitudinal layer are distinguishable. Villi are minute projections of the mucous surface, barely visible to the naked eye. They impart a velvety appearance to the inner surface of the small intestine. They are less numerous in the ileum than the jejunum. They contain from one to three arteries, veins, [and a lacteal.

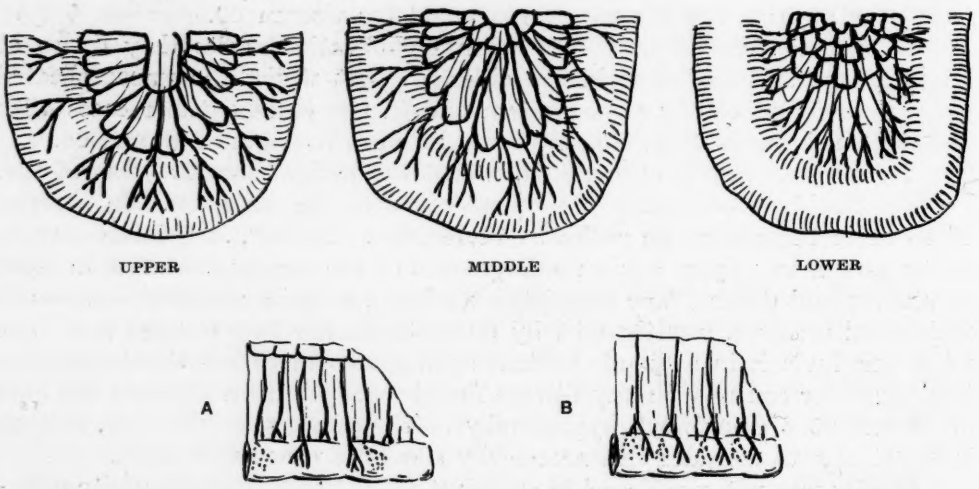

FIG. 118.—To illustrate distribution of blood-vessels to the small intestine, upper, middle, and lower portions. A, Peripheral terminations as actually found; B, As described in text-books.

The contents of the latter are propelled along by an involuntary muscle derived from the submucous coat.

The valvulæ conniventes are transverse folds extending partially around the gut. They are never obliterated even with distension. They are much larger on the attached side of the gut than on the free one. In the latter site they are often entirely absent, as also in the lower ileum.

The glands of Lieberkühn are simple tubular depressions found not only in the small but in the large intestine also. They are closely set, narrow, and extend through the thickness of the mucous coat as far as its muscular layer.

Lymphoid tissue is scattered in solitary nodules or in considerable masses as Peyer's patches, found especially in the lower ileum.

The submucous coat contains blood- and lymph-vessels and the nerve plexus of Meissner. It is lax, but not sufficiently so to allow displacement of the valvulæ conniventes.

The muscular coat is composed of two layers. The inner circular is two to three times as thick as the outer longitudinal layer. The thin longitudinal layer, thickest at the free border, is *often imperfect, especially at the attachment of the mesentery.* The entire muscular coat diminishes in thickness from above downwards. The serous coat covers the intestine except at the point of attachment of the mesentery, where a bare area is left to allow blood-vessels to reach the gut. The bare area depends upon the amount of fat separating the leaves of the mesentery.

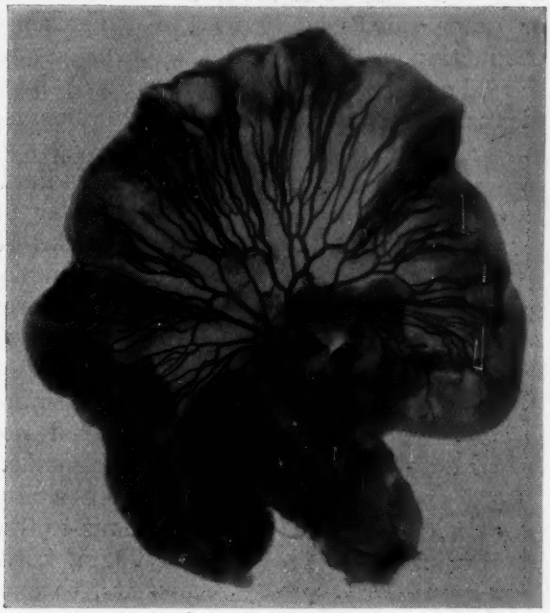

FIG. 119.—Arteries supplying portion of upper jejunum have been injected to show distribution.

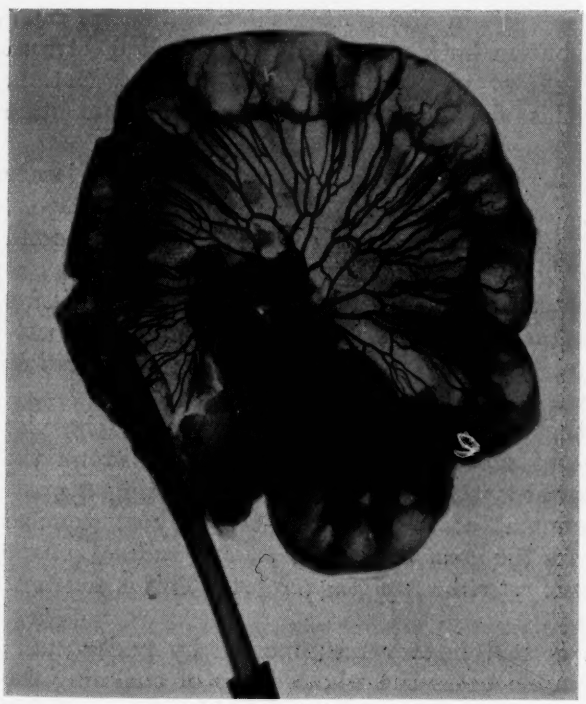

FIG. 120.—Similar to *Fig.* 119, but the intestine in this case is distended with oxygen.

The blood-vessels supplying the gut (*Figs.* 118–120) are always drawn in an artistic manner as forming a series of arcades (*Fig.* 118, B) from which terminal branches are given off, which would seem to bifurcate and to straddle the gut (as a rider would his horse), giving symmetrical branches to each side of the gut. Such in real life is not the case. The terminal artery leaves the arcade at a distance from the gut and passes to one side of it, and the next artery supplies the opposite side farther along, and so forth (*Fig.* 118, A). Frequently between them a small artery will be seen to supply the mesenteric edge.

Such an arrangement helps to explain the position

that diverticula take when once they are produced. They tend at first to bulge into the mesentery, but later fall over the intestine (*Fig.* 121) to one or other side, usually to the same side as the artery which causes their production.

The nerves supplying the small intestine are derived from the solar plexus, and consist of medullated and non-medullated fibres from the central nervous and sympathetic systems. After piercing the longitudinal muscle they help to form the intramuscular plexus of Auerbach consisting of both varieties of fibres and also microscopic sympathetic ganglia. The nerves continue obliquely through the circular muscle layer and form within the submucous coat the plexus of Meissner. From this latter non-medullated fibres enter the mucous membrane and form periglandular and subepithelial networks as well as supplying the muscular tissue.

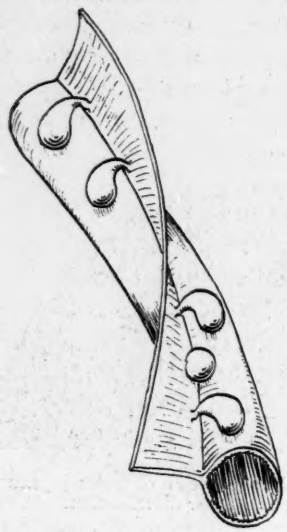

FIG. 121.—Diagram to illustrate diverticula on both sides of gut and into mesentery.

From a study of the anatomical features one is anxious to find what difference exists in jejunum and ileum, both as regards structure of wall and contents, to explain the frequency of these diverticula in the upper jejunum as compared with their paucity in the ileum.

Regarding contents there is little difference; in the jejunum the contents are somewhat more fluid, but in both places it is a fluid slimy stream which flows along. Regarding gas production, without obstruction and stasis this should not exist in the small intestine to any extent.

Comparing the lumina, one finds that the jejunum is three times larger than the ileum. Such decrease in diameter adds to the strength of the latter. It means that the same amount of longitudinal muscle fibres have to surround a smaller tube and therefore can give it a thicker coat.

In the jejunum in many cases the *longitudinal muscle is almost missing and certainly divaricated along the mesenteric border, and naturally in cases with much deposit of fat between the leaves of the mesentery this divarication will be greater still.*

Although the contents in the jejunum are greater in bulk and heavier in weight than in the ileum—two factors that would help to distend the gut and also to make it sag towards the pelvis, naturally exerting a great strain and pull upon the attachment of the arteries—the only anatomical factors worthy of consideration are the *great thinness of the longitudinal muscle* and the *greater size of the individual arteries supplying the gut.* This is constant and well marked.

The already weak portion is still further unsupported by peritoneum; thus, as a result, the circular muscle bears the whole stress of enclosing the bowel, and at the point where it is pierced by the artery with accompanying vein, fatty sheath, etc., a point of natural weakened resistance is produced.

PATHOLOGY OF DIVERTICULA.

The diverticula are thin-walled sacs. A section of the wall at the fundus of the sac consists of the mucous membrane of the intestine covered by the peritoneum forming one of the leaves of the mesentery. Between the two there may be some areolar tissue and fibrous tissue (*Fig.* 122). In most cases there is a marked deposit of fat encasing the sac—a similar finding to diverticula in the sigmoid. The presence of such fat is a difficult problem. Is it a primary condition and one of the factors concerned in the production of the disease? Has it, by increasing in amount, separated the mesentery, which separation in turn has caused a longitudinal divarication of the outer longitudinal muscle of the gut?

If fat is a primary condition, then why has the jejunum been affected where a deposit of fat is rare, while the ileum, where the mesentery is normally loaded with tabs of fat, is usually unaffected? Is the presence of this fat a precursor of hernia, just as it is in the inguinal and femoral regions, where, by dilating the canal and by subsequent traction, a track is formed for the future peritoneal protrusion? Again, in the heart, a diverticulum of the ventricle is subsequent to a heavy deposit in most cases, not only on the surface, but also leading between the fibres of the cardiac muscle. In diverticulum of the bladder and œsophagus the sac is usually encased with a thick deposit of adipose tissue. Or again, is the fat entirely secondary? Every surgeon has been impressed by the heavy deposit of granular fat around a pyonephrotic kidney. At the moment a gall-bladder is condemned because of opacity in its walls. This fat is said to represent past infection.

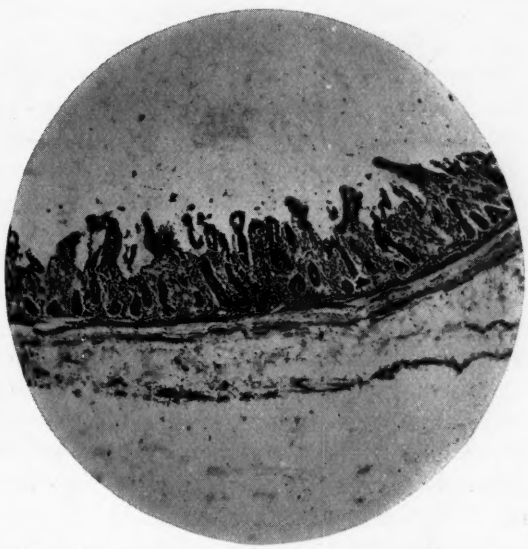

FIG. 122.—Low power. Section of fundus of diverticulum. Note absence of intestinal muscle, but muscularis mucosæ is still present.

Examples could be multiplied. No definite conclusion can be reached upon this point except that the character of the fat in those cases where it is primary is that of true adipose tissue—large clear cells with the nucleus in its typical circumferential position, compared with the granular fat found in the secondary cases, where a rich deposit of leucocytes and small round cells gives the fat a more elastic and resilient feel.

As has been pointed out, so many of the false diverticula have been without signs of inflammation that no cause has been given for the formation of a secondary deposit of fat.

Between the serosa and mucosa a variable amount of fibrous tissue may

be found. Its presence is a direct indication of inflammation in the sac, and as a result the serosa no longer moves freely upon the mucous membrane and a general hardness can be felt.

The origin of the sac is a projection, herniation, or protrusion of the mucous membrane at the mesenteric border (*Fig.* 123) of the gut through the substance of the muscle. As shown in the anatomy of the small intestine, the longitudinal muscle is strongest along the antimesenteric border, where peristalsis is greatest, and least along the mesenteric border. In addition, this area is unsupported by peritoneum, and if, by the deposition of fat, as is usual, the bare area is further increased and the muscle further weakened, either by pressure atrophy or by its fibres being divaricated through the fat passing inwards, additional cause will be found for herniation.

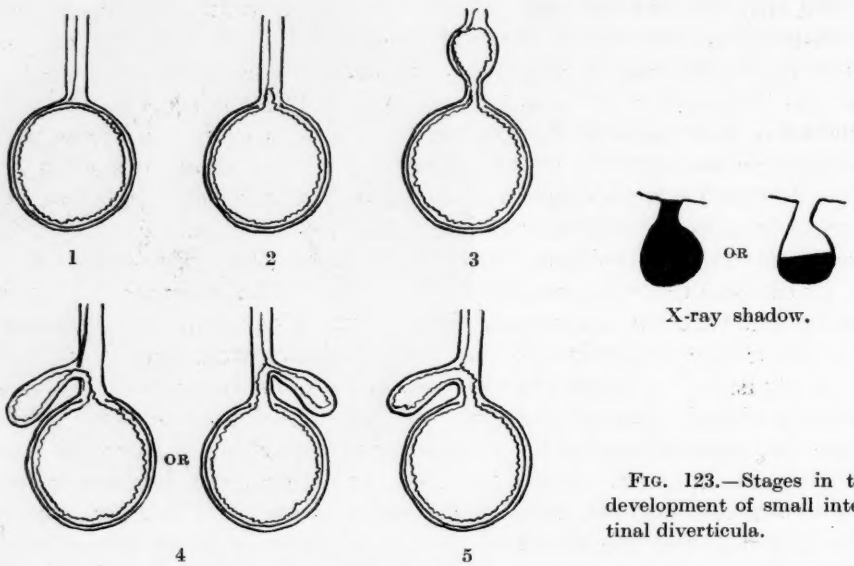

FIG. 123.—Stages in the development of small intestinal diverticula.

Such causes explain the weakness in the longitudinal muscle, which admittedly has weak points, but the circular muscle, which normally surrounds the bowel with an equal distribution of its fibres, must also be weakened. In this respect there is some definite relationship with the artery, because the sac occurs at the point of penetration of the artery, as can be seen in *Fig.* 133. Either a channel exists at the point of entry of artery and vein, and through this the mucous membrane bulges, in which case of course the circular coat would be pushed aside—in fact there would be a condensation of circular muscle on either side of the neck of the sac—or the pull of the artery on the wall or the drag of heavily laden ptosed bowel away from the fixed artery would pull out a conical tube of bowel, in which case it would contain all the layers.

In *Fig.* 122 it will be seen that no muscle exists, showing that it is a genuine pressure rather than a traction diverticulum. Confusion should not arise over the circular and longitudinal muscle and the muscularis mucosæ. It is clear that even in false diverticula the muscularis mucosæ will be present.

In the larger sacs an occasional strand of muscle can be seen. This is only found in the cases where two separate diverticula have become fused, and the small band of circular muscle which previously lay between and separated them is carried over the top as a thin band, causing a dimple at the tip of the sac and making the fundus, instead of globular, somewhat heart-shaped (*see Fig.* 125).

That such fusion takes place is well recognized. In Boling's case, at the first operation the gut was seen to be studded with 250 to 300 diverticula, but at a second operation, owing to fusion only 53 were present although individually the average size was greater. Naturally the strap of muscle between would now be represented upon the sac as an atrophied band. At first appearance the specimen might be mistaken for a case of pneumatosis of the small intestine. This rare condition, described by Nitch and later by Pugh, includes the presence of air sacs—large and small—in the mesentery, either close to the wall of the gut or passing towards the root of the mesentery, and gives the feel of surgical emphysema. The causative force is not known; possibly it is a sequela of aerophagy. There is no connection, however, between pneumatosis of the mesentery and false diverticulosis.

In size the sacs vary—anything from a pea to a small orange or apple. In the writer's case the average size was that of a golf ball. The projection, although always on the mesenteric side, is not absolutely mid-line. As the sac enlarges it grows between the layers of the mesentery, but sooner or later tends to bulge to one or other side (*see Fig.* 121). This is either: (1) Because the sac is following the particular artery which has been the determining factor in its production, and this may be on one or other leaf of the mesentery; or (2) For purely mechanical reasons, as it is easier to accommodate the growing sacs in a given area, if they take up a position alternately on either side of the bowel instead of being side by side. In Berry's case the majority fell to the right side of the gut. He considered that owing to the obliquity of the mesentery there was greater room on the right side than on the left to accommodate them.

Mention has been made of the relationship of the sacs to the straight terminal arteries which supply the bowel arising from the terminal arcade. The relationship of these vessels is definite and constant and is the only incontestible factor in the etiology of the condition. Hensemann was the first to point this out, and proved his point by serial sections.

The sacs in the uncomplicated cases are empty or contain some fluid chyme intermixed with air. Inspissation of the contents is rare for many reasons—the contents are very fluid, stasis is rare, the stoma is large so that ingress and egress to contents is easy, peristalsis in the small intestine is rapid, etc. In the literature the presence of inspissated 'fæces', gall-stones, and malignant growths is mentioned in individual and isolated cases, but judging by the large proportion of sacs in individual instances—up to 500 in some—and the number of cases without symptoms, it can be readily realized that stasis and other complications in these sacs are relatively rare.

Naturally complications do arise from time to time, probably owing to the enlarging of the sac and to the narrowing of the neck. In the early stage the sac is small and the stoma is large in proportion. As long as the sac

lies in the mesentery, naturally the opening into the bowel will be at its lowest point, and with the patient upright there will be good gravity or dependent drainage. Later, when the sac hangs over the edge of the gut, the neck gets lengthened out and narrowed, drainage is uphill, and stasis will naturally ensue. A typical example of fæcal stasis is shown in Watson's case. This complication is the exception in small-intestine diverticula but almost the rule in the colon.

Hirschmann reports a case of cancer starting in the diverticulum, and Holt a case where a sarcoma and diverticulum are associated. There is no definite evidence to show which is the primary factor. Diverticulitis and peridiverticulitis are the usual complications leading to localized or generalized infection. Kinking and obstruction are very apt to ensue in such cases, e.g., those of Gordinier and Sampson, Braithwaite, Kaspar, the writer, and others. Infection around the diverticulum is more frequent in the colon or duodenum. Molesworth reports in this connection an interesting case where there was associated with the duodenal diverticula considerable diverticulosis of the sigmoid. The patient died of pancreatic necrosis secondary to inflammation of the duodenal diverticulum.

The emptying of the sacs is dependent upon two factors: (1) gravity, and (2) the contraction and approximation of the leaves of the mesentery. This last factor is only free to act as long as the sac is in the mesentery, as in the early stages. But later when it protrudes through the anterior or posterior layer (*see Fig.* 123) it will no longer be so compressed. The question of fat again arises. The sac can only be compressed if the two leaves of the mesentery can be approximated, and this naturally is impossible if a thick layer of fat is sandwiched between.

The absence of muscle in the wall of these sacs means that they are unaffected by the general peristalsis of the gut. The suction or *vis a fronte* of the stream as it flows past may empty them, but they have no driving force or *vis a tergo* of their own. The absence of muscle in the wall also results in the absence of colic, which is a marked feature of all 'true' diverticula.

In the early stages gravity is sufficient, but later, with kinking of the neck, drainage is rendered more difficult and a feeling of fullness and distension exists, and if no adhesions are present the sac can only empty by the patient's changing from the standing to the lying position. This explains the comfort experienced in many cases—the writer's patient invariably got relieved of this distension by lying down. With occlusion of the neck, stasis, putrefaction, and gas production are natural sequelæ, clinically manifested by borborygmi, distension, and flatulence.

Regarding the situation of the diverticula, they may arise at any point from the duodenojejunal angle to the ileum. When arising at the flexure they cause trouble both as regards diagnosis, in that they often simulate a Haudek's niche (Hurst, *Guy's Hospital Reports*, 1932, March—*see Fig.* 130), and also in their surgical treatment. The largest of the sacs are usually at the proximal portion, but this rule is not absolute, as often one finds at the beginning a series of large sacs followed by smaller ones, but scattered through the latter at irregular intervals many large ones.

The number is never constant. It may vary from quite a few to perhaps 500 to 600. There is nothing constant about the cases which give rise to symptoms. They may arise in diverticula near the duodenojejunal flexure or in ones farther down, and may occur in patients with few diverticula as readily as those in whom the small intestine is just studded. They do seem to occur more readily in the giant diverticula than in the small ones. The sac wall is generally very thin. At operation the fundus can be invaginated with the finger right into the lumen of the intestine with great ease.

THEORIES OF CAUSATION.

For many years it was thought sufficient to classify diverticula into congenital and acquired, or true and false, the latter terms depending upon the presence or absence of muscle in the wall of the sac.

Kausch was the first to point out that 'congenital' and 'true' and 'acquired' and 'false' were not synonymous terms. Brandes reported a case of acquired diverticula which were 'true' in that all the wall was present. At an early stage it was realized that these diverticula were an acquired condition for many reasons: (1) never found in the fœtus, (2) never found in the young, (3) a disease of the retrogressive period of life, (4) incompatible with embryological growth, (5) study of the progress of the disease within the same patient, e.g., Boling, Stevenson, et al., (6) their structure, (7) their inconstancy in size, shape, appearance, situation, (8) absence of other congenital defects, etc.—in fact, no brief could be held in favour of the congenital theory. On the acceptance of the disease as an acquired condition many theories have been produced to explain its occurrence.

Roth in 1872 formulated the idea that it was a fatty degeneration of the tunica muscularis. But why should it attack jejunum rather than ileum? The patients are not necessarily obese, some are young and healthy (the writer's case, age 30). Histologically there is no proof that the longitudinal muscle in the upper jejunum has alone been involved in a fatty degeneration.

Klebs in 1869 pointed out that there was an artery in relation to the sac. He was of the opinion that the cause was the drag of a heavy gut away from the fixed vessels, with the formation of traction diverticula. Such diverticula are all true and contain all the layers of the gut. It is obvious that a sac that falls over the edge of the gut cannot have an artery exerting tension upon its fundus.

Hansemann in 1899 examined carefully the relationship of the artery to the sac, and by serial sections was able to show that the pouch appeared *beside* the artery, and that, although the artery did not draw out the sac, the sac made use of the channel so formed to bulge through the muscle. He considered the causative force was increased intestinal pressure from gas or fæces. In support of his thesis he produced diverticula by inflation.

Chlumsky in 1899 repeated Hansemann's experiments, and dilating the gut found it ruptured at the antimesenteric border, and concluded that the condition was not due to increased intestinal tension.

Della Palma Modesto blew air into gut deprived of its peritoneum. In one case only did a diverticulum appear, and in this the patient had died of

tuberculosis. He concluded, therefore, that this disease is only produced in the intestine of people suffering from some degenerative disease, e.g., tuberculosis or old age, or else that there is a congenital predisposition.

Gisbertz in 1929 mentions this fact when he reports the case upon which he operated under local anæsthesia. The patient was suffering from tuberculosis of the lungs and glands of the abdomen. He holds that each gut has a tendency to form diverticula and each intestine is a law unto itself. The appearance of a sac depends upon the hole in the wall through which the vessels pass. He believes that external circumstances are the exciting cause: occupation, mode of life—in fact anything which modifies the peristalsis of the gut.

Graser (1899) thinks that venous congestion, e.g., portal obstruction, is the deciding factor. The distended veins, by enlarging the opening in the muscle, produce a *locus minoris resistentiæ* and, as a result, hernia formation. He found supporters in Akerlund, Oehnel, Moreau and Mardock, Della Palma Modesto, Walanaba, Buchni, et al. They held that the disease occurred almost exclusively after the age of 40; it was the period of circulatory embarrassment and failure.

Sudsiki in 1900 produced the theory that, rather than the vein actively distending, it was allowed to do so passively by the diminished resistance of the connective tissue.

Davies-Colley considered that the fat around the vessels and between the muscle fibres was the cause. It acted by causing a weak spot in the muscular tunic, or by its pressure causing the muscle to atrophy, or by infiltrating the muscle reducing its resisting power. This helps to explain its position in the mesenteric border in the small intestine and in the region of the epiploic bodies in the large. Such a theory is not tenable. Fat is much more heavily present in the ileum. It is well recognized that the epiploic bodies have no part in the causation of sigmoid diverticula. They contain the diverticula because they contain the artery, and the artery is the conductive force determining the direction that the diverticula will take.

That 'atrophy of muscle' must play an important part is stressed by many, and Neupert reporting upon a case considered 'muscle weakness' to be the primary cause. Boling in his article considers lack of 'muscle tone' the most vital factor.

Traction diverticula arising from tumours, myomata, hæmangiomata, lipomata, tuberculous glands, etc., are frequently quoted, but no serious attempt can he made to correlate such cases with the condition under investigation.

General cachectic states resultant upon tuberculosis, typhus, chronic osteo-arthritis of the spine, carious teeth, etc., are said to be more prevalent in those with diverticulitis than in an equal number of individuals of the same age used as controls.

Although Keibel and Mall and Lewis and Thyng have shown the presence in the very early fœtus of islands of isolated cells which normally atrophy, but which by persisting have produced cysts and diverticula, no connection can be claimed between them and the diverticula under examination at the moment. Such diverticula or cysts are included under congenital diverticula of enterogenous origin.

Nitch described in detail the condition of cystic pneumatosis of the mesentery in which there are localized 'gas bags' in the mesentery. This, although interesting and clinically somewhat similar in appearance to multiple diverticula, cannot be considered as an etiological factor.

In 1923 Helvestine, discussing the condition, concluded that no single factor could explain the disease, and decided that the three following factors working together were necessary: (1) Traction by mesenteric vessels or following adhesions; (2) Degeneration of intestinal musculature; (3) Increased intra-abdominal pressure. One cannot agree with this:—

1. Firstly, adhesions can be shown to be absent in all sacs unless inflammation has taken place, and in such cases the adhesions have an inhibiting influence rather than otherwise upon the growth of the sac. Traction by mesenteric vessels does not occur. If so, one would expect to find a vessel attached to the fundus of the sac and the artery taut; also the fundus of the sac would consist of all the coats and not mucosa only.

2. Degeneration of intestinal musculature: Why should this involve the longitudinal muscle only? Why only that portion in the mesentery? Why only in the jejunum and not in the ileum?

3. Intra-abdominal pressure: Such probably was considered as a possible cause from the fact that many of the patients had died of enlarged prostate with probably vesical tenesmus, many had inguinal herniæ, and many were constipated. It has, however, been pointed out on the other hand that many had lax patulous abdomens, and, as in the writer's case—a woman of 30—little straining of any sort could be proved.

Sheppe modified Helvestine's views slightly and gave as the three factors: (1) Rectal or vesical tenesmus; (2) Atrophy of inner circular layer of intestinal musculature; (3) Traction of mesenteric vessels due to sclerosis and adhesions. Of these, the second is especially worthy of note. Degeneration of the circular muscle would surely produce a circular bulge involving the circumference of the gut over the area involved, and could in no way explain a series of bulges involving the gut in its longitudinal direction.

Any theory propounded to explain the origin of these diverticula must, if correct, embrace the following facts: The disease occurs in both sexes, usually after 40, but it may be found in patients as young as 30; the upper jejunum is usually affected and the larger diverticula are seen nearer the duodenum; there is a definite relationship with the artery, and in several cases a stricture further down.

A fact overlooked by all writers, and one which seems of paramount importance, is the *anatomical disposition of the longitudinal muscle in the upper jejunum*. It is thinned out, wasted, or almost missing owing to the fact that the jejunum has a three times greater circumference to be covered than the ileum. In fact, one might almost say that just as the colon has three longitudinal tæniæ, so the jejunum has longitudinal tæniæ on three sides only. This congenital divarication can be still further accentuated by separation of the mesenterial leaves by fat. The circular tunic is the only one left to protect the gut, and it in turn is pierced by the artery and veins to the gut, giving a potential seat of lowered resistance. In addition the individual arteries to the jejunum are larger than those to the ileum. The further

factor needed is increased pressure such as is found in stricture of the gut (Godard, Jefferson, Cooper), constipation, vesical straining, coughing, etc.

Experimentally the production of diverticula occurs at these situations by increased intra-intestinal pressure, and the hernia so produced is a false one analagous to the sac found clinically.

One must conclude that: (1) Increased intestinal tension, (2) acting along the channel for the artery, (3) at the point where the longitudinal muscle is divaricated, is the causative agent.

CASE REPORT.

The following personal case helps to exemplify the condition.

Mary B., a spinster of 30 years of age and a shop assistant until three years previously, was sent from the country to hospital with a diagnosis of perforated peptic ulcer. Seen soon after arrival her abdomen was tender in the epigastrium and she had vomited; but beyond slight elevation in pulse and temperature nothing definite could be discovered. Her abdomen was not rigid. Liver dullness was present. It was fairly definite that she had not had a perforated ulcer, and as she had a history of three previous attacks it was decided to wait.

The following day she had improved greatly and had an opaque meal examination which showed " multiple obstructions in the small intestine, ? Adhesions, ? T. B. peritonitis ". Two days later she developed acute complete small-intestine obstruction, and she was operated upon at once. The upper jejunum laden with fat was studded on the mesenteric border with large diverticula; some lay between the layers of the mesentery, but others fell over the edge of the jejunum, some on one side and some on the other.

FIG. 124.—Sketch of bowel resected. Seat of perforation is easily seen.

At one point a hard mass was found in the mesentery from which pus was escaping, and at that point the gut was angulated and obstructed. Rapidly and carefully to prevent peritoneal soiling the writer excised 4 ft. of the small intestine about 6 in. from the duodenojejunal flexure, and including, it was thought, all the affected gut. At the time further examination of the abdomen was impossible as the intestine had to be packed away with meticulous care. Although at the time it was thought that all the diverticula had been removed, subsequent radiograms showed that this was not the case. The patient made an uneventful recovery and since has remained perfectly well.

The most noticeable features were the enormous deposit of fat, the thinness of the walls of the sacs—with the finger the fundus of each could be invaginated with ease into the lumen of the gut—their enormous number, their individually large size (average was that of a golf-ball), and the relative absence of grave symptoms previously. There were signs of old scars, and from her symptoms it would appear that she had had either three previous attacks of peridiverticulitis without perforation which had settled down, *or* perforation with localized abscess, which had drained into the gut and had not given rise to adhesions and obstruction as on the present occasion.

Fig. 124 gives an idea of the macroscopic appearance of the bowel removed at operation. The excised intestine was filled with bismuth solution, and the

radiogram taken (*Fig.* 125) gives an exact picture of the relationship of diverticula to jejunum. It will be noticed that the small diverticula have a round fundus, the large ones are depressed and dimpled owing to the strap of circular muscle mentioned previously. The resected specimen measured 4 ft. in length, and the upper end started 6 in. from the duodenal flexure. There was sufficient tissue to allow a satisfactory side-to-side anastomosis to be performed.

FIG. 125.—Radiogram of resected specimen filled with barium. Note diverticula present along the concavity —mesenteric border. A, Perforation.

Fig. 125, A shows the point of perforation of the diverticulum. Since several of the diverticula lay behind the intestine, the outline is masked by the bismuth in the latter. During the convalescent period further X-rays were taken and it was rather a surprise to find further diverticula still present. With the co-operation of a willing patient a series of these was taken hourly for ten hours, and, as a result the appearance of diverticula at different times and in different stages of emptying has been obtained (*Figs.* 126–128). Some of these show the typical pyriform or globular mass hanging from the intestine. In others, where the sac contains the bismuth below with gas above, there is the saucer-shaped shadow with a more or less horizontal upper surface.

The patient's previous history showed her to be a strong healthy girl up to three years ago—fond of tennis, rowing, walking, etc. Since then she had had intermittent ill health; this included a thyroidectomy two years ago.

FIG. 126.—Five-and-a-half-hour picture. Globular shadows distinctly seen.

She complained of upper abdominal pain, pain after food, and even before food, though pain was occasionally eased by light food. The pain did not radiate and it had recently become almost constant. Its usual situation was slightly above and to the left of the umbilicus. On a few occasions she had had night pain, and had given up meat, potatoes, and heavy food. Her pain suggested in an indefinite way a peptic ulcer. There was no vomiting as a rule, but flatulence and borborygmi were a marked feature. From being a strong healthy girl she had been forced to give up work altogether.

During this period of three years she had had three very acute attacks. In the first, which was diagnosed 'acute gastritis', the vomiting was so excessive that stomach lavage was resorted to upon several occasions. The second attack was diagnosed 'peritonitis'

FIG. 127.—Note three diverticula hanging from intestine (not filled). This was taken four and a half hours after ingestion of the barium meal.

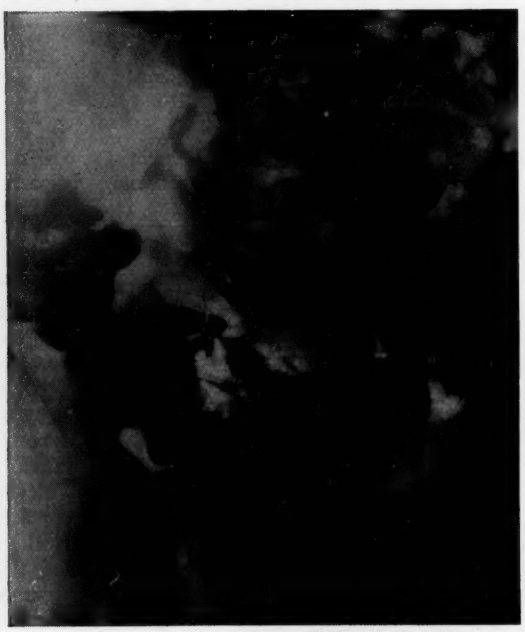

FIG. 128.—Ten-and-a-half-hour picture. A diverticulum can be seen in right-hand upper quadrant of radiogram opaque in lower half, gas in upper portion.

—sudden severe abdominal pain, with abdominal rigidity, etc. She was carried home and kept in bed for two months. The third attack was like the second; she was admitted to hospital and prepared for operation, but as the attack subsided, operation was postponed and she was eventually discharged.

On physical examination there was little of interest. The abdomen moved freely; there was general tenderness and muscle guarding, but no tumour and no free fluid. Liver, kidney, and spleen were not obviously enlarged. There was no rigidity. Rectal and vaginal examination were negative. In fact, from her symptoms and signs no definite conclusion could be reached.

SYMPTOMS.

It has been found impossible to draw up an exact clinical picture of these cases with exact text-book signs and symptoms. Such naturally is the case in a disease which at the one extreme may be symptomless, and at the other may be producing symptoms of the severity of acute intestinal obstruction. In the following description of the symptoms an attempt has been made to collect and correlate any feature that seems common to all the cases so far recorded in the literature.

Duodenal diverticula are not so serious as colonic, and jejuno-ileal diverticula are much less serious than either. Just as these three areas vary in the gravity of their prognosis, so one finds the vagueness of their symptoms bearing a direct relationship. Fully three-quarters of all the recorded cases were without symptoms, and in the remaining quarter the symptoms varied greatly, depending upon whether the patient had a long period of chronic abdominal ill health or was first seen as a surgical emergency.

Symptoms depend upon closure of the neck with resulting inflammation and the production of a closed cyst. In the chronic case the symptoms resemble a peptic ulcer in a general way, but, as Frank Smithies pointed out in 1920, there is no clear-cut dyspepsia and certainly no periods of remission.

Pain is a marked feature, but never very severe, never colicky in character like Struther's case, where the diverticulum had a muscular wall, and, as a result, was capable of producing a pain of intense severity. The pain is chiefly epigastric, and passes to the left of the umbilicus and even down the left side of the abdomen. It varies with the situation, and is due to distension of the sacs. Case, operating upon a patient under local anæsthesia, was able by compressing the distended sacs to produce a pain which the patient agreed was similar to the original one. Pain in the early stage comes on half an hour to three hours after food, but in the advanced stages it is present almost all the time. Food, fluids, or alkalis have little or no effect upon the pain. Greatest comfort is obtained by lying down.

Pain is greatly aggravated by coarse food, potatoes, and acid meals. In this respect these cases greatly resemble the series of 34 duodenal diverticula described by Oehnel and Akerlund. Such also is found in duodenal ulcer, and several patients have had a history of fifteen to sixteen years with the pain worse after meals, and with a radiation to the shoulder. Shoulder-pain is not a feature, but many of the cases are complicated by an additional disease, e.g., gall-bladder trouble, etc., the symptoms from which may tend to mask the original condition.

Probably the most constant and most interesting feature of the disease is the flatulence, borborygmi, gurgling, and rumbling that all suffer from. In Usland's case the patient described his abdominal sensation as being like 'clothes boiling in a pot', and in Braithwaite's case the patient had to retire to prevent his confusion. All complain of distension, and this was much greater with standing than lying, and was eased usually by eructation of foul gas or sour fluid. The meteorism and weird noises, being such a feature, are one of the helpful points in differentiating the disease from a peptic ulcer. The age factor must also be considered. The ulcer history has usually started before 40, whilst diverticula are rare till considerably after that. Effect of alkalis and periods of remission also help, but in most cases diagnosis is difficult, and when the two diseases are coexistent, as is not infrequent, a definite diagnosis clinically is impossible.

Vomiting is not constant—it was a marked feature in some cases, e.g., those of Rothschild and Case, but variable with others. Nausea is more frequently found. Constipation is the rule rather than diarrhœa, as compared again with duodenal ulcer, where the hypermotility in the colon usually ensures a regular movement of the bowels. Two cases vomited blood, but this is not mentioned in the others. In one diarrhœa and constipation alternated. 'Mild prolonged indigestion', 'bilious attacks', 'attacks of auto-intoxication', loss of appetite, coated tongue, muddy complexion, and general debility, are mentioned in many of the cases.

Loss of weight is quoted by some as an important feature, e.g., Hunt and Cook's patient had lost 3 stone. MacKechnie's patient was very emaciated, but others, e.g., Rothschild, point out specifically no loss of weight, and the writer's case had actually gained. One feels that any change in weight in these cases is not the result of the disease, but depends upon the liberality or otherwise of the diet permitted.

That melæna is a marked feature is stressed greatly by Continental writers, especially the Scandinavians. The patients of Hunt and Cook, Rothschild, and MacKechnie all showed this condition. Usland in his case was able to show a venous plexus in one of the sacs actually bleeding at the time of operation. The bleeding was very severe in Hartung's patient.

Great care is required to separate the symptoms of the diverticula proper from those of some other coexisting disease. In 1921 Terry and Mugler collected 19 cases of jejunal diverticula. Of these one occurred at the duodenojejunal flexure, but in only 3 were the symptoms attributable to the diverticula.

The fractional test-meal is reported by Oehnel to have failed as a clinical aid to a diagnosis, but as any one writer gets so few examples of such a rare disease, no sufficient series can be obtained on which to form a definite estimate of its value.

There is no constancy in the coexisting diseases. Gisbertz' case had tuberculosis of the lungs and abdominal glands; many of the early cases had uræmia, others duodenal ulcer, gall-stones, etc., so that no definite causal relationship could be established.

The above series of symptoms merely accentuate the fact that no clinical syndrome is known to include all the cases. The very vagueness of the condition is one reason for adding to its interest.

RADIOLOGY.

In 1914 Forssell (radiologist) discovered by barium meal, and Key (surgeon) confirmed by operation, the presence of a duodenal diverticulum. This was the first recorded case of a definite pre-operative diagnosis in the history of the disease. It was not till 1920 that Case (Battle Creek) was able to find the first jejunal diverticulum and to confirm it by operation. In an excellent article he described 5 such cases. The radiological evidence cannot be doubted, but only 2 had subsequently laparatomy to verify their presence.

In the somewhat similar cases of Boling and Stevenson, the abdomen was opened and intestines with 150 to 250 diverticula were seen ; no resection was performed, and yet in both cases no sign of the diverticula appeared radiographically although repeated X-rays were taken with the knowledge that the diverticula were present. Braithwaite and Berry had positive results in their cases only after difficulty. Heidecker in 1926, and Tengwall in 1930, also claim positive X-ray results, the latter only after repeated pictures had given negative results.

In 1924 Baastrup reported a case where the diagnosis had been made after X-ray examination. His case, although interesting, was a single traction diverticulum due to the presence of softening tuberculous glands, and as such should not be included under multiple false diverticula.

Gisbertz in 1929 reported an interesting case. The operation, although performed with great care under local anæsthesia, was unsuccessful. The chief interest lay in the pre-operative X-ray diagnosis. C. Orbaan also records in a Dutch paper a case diagnosed radiologically. Such a small list makes one realize either how difficult the cases are to radiograph or how necessary is some further improvement in technique.

In the writer's case the shadows seen in the pre-operative picture were recognized only *after* operation as diverticula, as the eye then knew what was to be expected. Incidentally the condition was unsuspected by the writer. In the series of photographs taken after the operation the shadows assume the typical appearance. This series is unique in that it shows diverticula at hourly intervals and in different degrees of fullness. Case, describing his radiograms says, " the findings present an interesting relief from the monotony of everyday life ".

Infinite patience on the part of the radiographer is required in taking the series of photographs, in timing the picture correctly, and in placing the patient at the correct angle. In reading the radiogram considerable skill and knowledge of the normal and an appreciation of super-position are required. A large meal by its bulk will obscure the picture and by its weight will drag the stomach down into the pelvis if the patient is standing, or into one or other of the paravertebral groves if the patient is supine. Hence a small meal.

A series of pictures at hourly intervals will show the constancy of a shadow, and if two films are superimposed, the fixity of the part can be seen. A twenty-four hour picture often will show the shadow still present ; the lack of muscle in the wall of the diverticulum naturally means very sluggish emptying.

A preliminary X-ray without bismuth is recommended in all cases in the hope that the small sacs will be seen filled with air or gas, and by the shape of the rounded margin a diagnosis be made. Such has never been successful. With a barium or bismuth meal the typical shadow of a diverticulum is either a round or pear-shaped shadow communicating with the gut. As the latter is usually empty, a series of such shadows side by side is typical (*see Fig.* 127).

The other form that the diverticulum may take is that of an opaque semicircle or D (with the straight line horizontal) and above this a similar shadow filled with air. The two portions are separated by a horizontal line, similar to what one would expect to find from a balloon partially filled with a heavy fluid below and gas above. *Fig.* 129 shows the condition diagramatically. When such a picture is positive a definite diagnosis is simple, but it is disheartening to see intestine studded with diverticula and yet with radiography to obtain a negative result each time.

FIG. 129.—To explain the saucer-shaped shadows. (*After Godard.*)

There are two or three possible explanations of this :—

1. The diverticula are still in the mesentery, i.e., attached to the *upper* margin of the gut, and hence do not fill ; filling in such cases could only be obtained by placing the patient in the Trendelenburg position.

2. The diverticula, either large or small, are *already filled* with some other medium which must first be displaced ; abdominal massage might help to fill the sacs. Such a possibility is rare, as at operation the sacs are usually entirely or partially empty.

3. The diverticula have become so large that, hanging down on either side of the gut, the neck is kinked and entrance is cut off. This also is unlikely, as the stoma will usually admit one finger and even two with ease.

The writer's case is an example of the disappointing results of X rays. *Fig.* 125 shows the 4 ft. of intestine removed and filled with bismuth, and yet *Fig.* 127, taken before operation, gives no indication of the presence of diverticula to such an extensive degree.

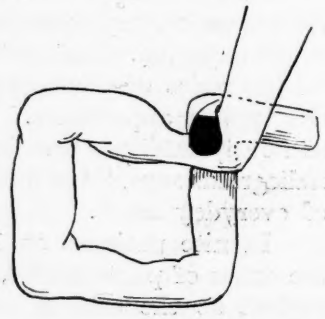

FIG. 130.—To illustrate the difficulty in differentiating the shadow of a Haudek's niche from that of a duodenojejunal flexure diverticulum. (*After Hurst.*)

With a diverticulum at the duodenojejunal flexure, difficulties arise. First, on an antero-posterior view the stomach, if filled with an opaque meal, may entirely mask the condition by covering up the smaller shadow. Secondly, a perforating gastric ulcer may produce a Haudek's niche almost identical in appearance to the duodenojejunal flexure shadow. This has been pointed out by De Quervain, by Akerlund at considerable length, and recently (1932) by Hurst in *Guy's Hospital Gazette* (*Fig.* 130). The first difficulty can be overcome by a true lateral or partially

lateral picture; but the second difficulty can only be overcome by screening the patient. A Haudek's niche fills at once from the stomach meal, a duodenal or duodenojejunal flexure diverticulum is only seen *when* the meal has passed through the pylorus. A further picture taken after emptying and even up to twenty-four hours may still show a shadow. This may happen in any of the conditions and is of no value in making a differential diagnosis.

It is interesting to note that just as jejunal diverticula is a rare condition, its X-ray diagnosis is very much rarer. In duodenal diverticula the reverse holds good. It is diagnosed much more frequently in the X-ray room than in the operating theatre.

The most frequent errors in reading the radiogram of jejunal diverticula consist in considering the condition one of: (1) Gastric ulcer with Haudek's niche; (2) Generalized adhesions—tuberculous or otherwise; (3) Ptosis.

TREATMENT.

It is generally agreed that in 75 per cent of all cases the disease is symptomless. It thus passes undiagnosed or even unsuspected and is only found fortuitously at a post-mortem examination. In such cases the question of treatment naturally does not arise. In the majority of the early recorded cases the patients had died in old age and had spent a life free from major abdominal discomfort. The ages in Reinhardt's three cases were 64, 84, and 86. In all three there never had been any digestive symptoms at all.

A more difficult problem is the case where the condition is discovered during laparotomy for some other disease, e.g., gall-stones (Rothschild) or duodenal ulcer (Usland), or where a diagnosis has been made during a routine intestinal opaque meal examination.

Take the latter case first. Is it fair to subject the patient, usually elderly, to an operation which is not easy to perform, which is attended with considerable risk, and which, when the abdomen is opened, it may be impossible to complete satisfactorily? When one realizes that the majority pass through life without complications and that complications when they do occur have rarely been fatal, one cannot hesitate to postpone operation, with the understanding from the patient that he will keep under regular supervision every six months and report progress if any.

The former case is different. The abdomen is open, and it is now a personal matter for the surgeon. With a relatively young patient, with a competent surgeon, with diverticula localized to a limited area of intestine, resection of the affected portion probably is the correct procedure. If this is successful a perfect result may be expected, but in all cases a careful examination of the intestine below must be made to exclude a stricture. Such is found to exist in many cases, e.g., Sir Astley Cooper's original case; Henri Godard discovered a contracted scar in the ileum, where the patient had had the intestine caught in a strangulated femoral hernia. Geoffrey Jefferson was forced to do a double enterectomy not only of the area affected with diverticula but also the constricted portion below.

In all cases where symptoms, even if mild, have definitely originated from the diverticula, operation is advisable. In at least two cases the

abdomen was opened when the symptoms were mild, and when the condition was discovered it was thought to be inoperable and nothing was done. In both of these the patient returned later with aggravated symptoms. Enterectomy was performed with complete success.

In Stevenson's case the patient returned after two years, and several feet of the intestine with 154 diverticula were removed, with complete cure. Boling's case was interesting in that at the first operation there appeared some 250 diverticula, but at the second operation when a successful enterectomy was performed there were found to be only 53. Obviously fusion and coalescence had taken place to reduce their number.

In all cases where the diverticula are causing symptoms, even mild, operation is advisable, as it is noticed that practically all the patients in whom a major calamity, such as perforation or intestinal obstruction, supervened later had had a definite previous history.

Regarding the operative measures available in the uncomplicated case, the technique of choice is complete removal of the affected area with restoration of the continuity of the gut. In the writer's case the junction was of the side-to-side variety, as it was considered that the stoma in an end-to-end union might, on healing, contract and produce a slight contraction ring or stricture. This probably would not be sufficient in the normal individual to cause symptoms, but in a patient with a tendency already to form diverticula it might be the starting-point for further trouble. This is probably a small point, as most of the cases where technique was stated had an end-to-end anastomosis and the result seems to have been excellent.

At the upper end difficulty often arises, as the jejunal diverticula may be represented at the duodenojejunal flexure by other diverticula and even the duodenum itself may be involved. As a result healthy intestine cannot be obtained. This difficulty has been overcome by Rothschild and others by performing a gastrojejunostomy and using the opening into the diverticulum to form the new stoma between stomach and jejunum.

In other cases, e.g., Braithwaite's, where resection was at first considered impossible, a short circuit was performed. This, like all operations devised for two purposes, was a failure. If the short circuit works well, the loop does not drain, and if the loop drains well, the short circuit does not function. In Braithwaite's case ultimately a resection was performed with good results. Minor operations, such as removal of individual sacs, pleating, infolding, strengthening of the wall, etc., are recommended; but when perhaps up to 500 sacs may be present, and where the appearance of the sac is really a result or symptom of the disease, such operations naturally will be left for the theoretical surgeon and for the text-books. Such treatment is indicated in the single diverticulum of the congenital variety, such as Meckel's, where amputation with infolding gives a permanent result.

The extent of small intestine that can be resected without affecting the health of the patient has not definitely been decided upon. It must be remembered that jejunum is more valuable than ileum on account of its absorbing surface. Adams estimated that 7 ft. of jejunum or 12 ft. of ileum could be removed without disturbing the patient materially. In 1910 Bremen in a man of 61 removed 17 ft. 9 in. of small intestine. The patient lived two and

a half years and died subsequently of marasmus due to failure of fat absorption. The normal average length of small intestine is 22 ft., but the extremes vary from 15 to 30 ft., so that the important factor is not how much is removed, but how much remains. In animal experiments it is found that in dogs 50 per cent can be removed with safety. In such cases it is found that villi and mucosa hypertrophy considerably. As a general rule in man, if more than two-fifths of the entire small gut is involved, resection may be considered impossible.

It is readily realizable that spontaneous cure or recovery is impossible in these cases which are due to the absence of muscle in the wall of the sac. With all mucous herniæ, where the distension of the gut and sac acts as a *vis a fronte* distending the sac, and the adhesions on the exterior act as a *vis a tergo* preventing recoil, the disease is progressive. Thus in the case of those patients in whom operation is refused or contra-indicated, some regular mode of life must be advocated. In many cases the symptoms closely resembled a duodenal ulcer, and the restricted, alkaline, carbohydrate diet with which they were treated seemed to suit. All complained that rough food, potatoes, and meals with a residue did not agree. Small meals and often are most satisfactory. For the flatulence and borborygmi, alkalis and carminatives are of no assistance, as the gas is lying in a pouch, cut off from the main current of the intestinal tract by the fact that the neck of the diverticulum is kinked with the patient in the upright position.

Rest for half to one hour with the patient horizontal gives good postural drainage. In the writer's case rest in bed was a certain cure. At first sight it would appear useless to consider diet in these cases, as presumably well-mixed chyme leaves the stomach soon after a meal, and in three hours should have entirely passed along. This is so in the healthy intestine, and gas formation—the result of putrefaction or cellulose digestion after a period of hours—has not had time to occur. In the patient with diverticula the food gets into the sac, and, with the neck of the sac kinked, exit is impossible, and with stasis after many hours the sequelæ are obvious.

The non-operative treatment should include :—

1. Rest after meals or at regular intervals during the day—prone or supine.

2. Small light nutritious meals, the ingredients to have as small a residue as possible.

3. Alkalis and bismuth will prevent a too acid and irritating medium coming in contact with the mucosa in the diverticula, already reddened and inflamed with stasis and bacterial infection.

4. Olive oil will act similarly to No. 3.

5. Charkaolin or charcoal in some of its forms will act in conjunction with the above and aid in gas absorption.

6. Abdominal binder or massage may be tried.

7. Gaseous and aerated drinks to be avoided, and fluids to be taken between meals.

8. Straining is to be avoided—urinary straining, straining at stool, heavy physical work, etc. It is noteworthy how many of the patients had enlarged prostate, herniæ, etc.

9. Belladonna or atropine is recommended on the assumption that spasm of the intestine exists which may compress the neck of the diverticula.

10. Intestinal antiseptics, e.g., dimol, etc., if believed in by the physician, may be given.

In the emergency case no clear-cut technique can be followed. Each case is a law unto itself and has to be treated upon its own individual merits.

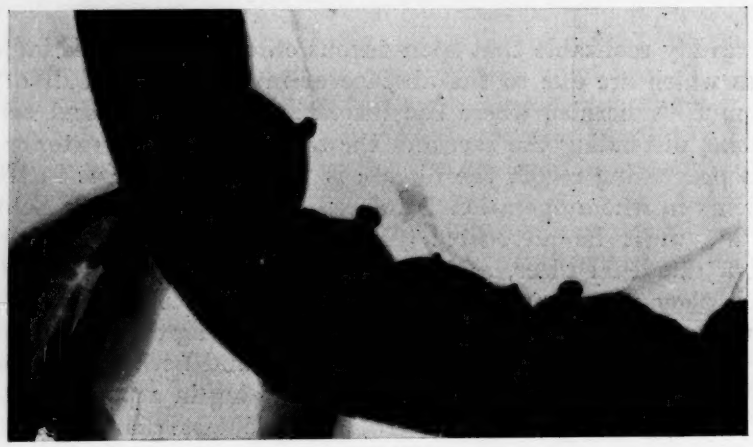

FIG. 131.—Gut and diverticula filled with bismuth solution.

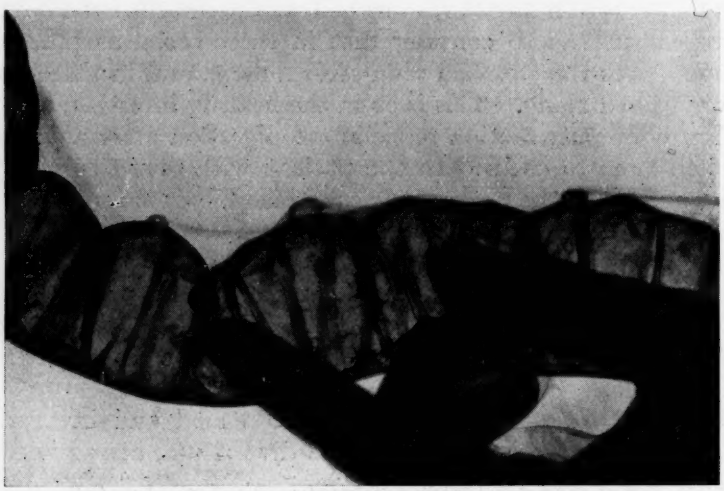

FIG. 132.—To show diverticula produced by distending jejunum with oxygen.

With perforation, with intestinal obstruction, with fistula formation, and with new growth, the super-added complication must first be treated and the disease proper dealt with later.

In conclusion, only one case in every four needs surgical treatment. The operation of choice is resection of the entire area unless too extensive. If this is impossible, short-circuit, drainage, or plastic operations must be attempted, although not as a rule attended with satisfactory results.

EXPERIMENTAL WORK.

The writer attempted to produce diverticula in the gut by distension alone. The material used was fresh post-mortem gut, and the distending medium was either a thin bismuth solution passed in under pressure by means of a Higginson's syringe, or else oxygen released gradually from a cylinder. With careful and gradual distension diverticula can be produced readily, and

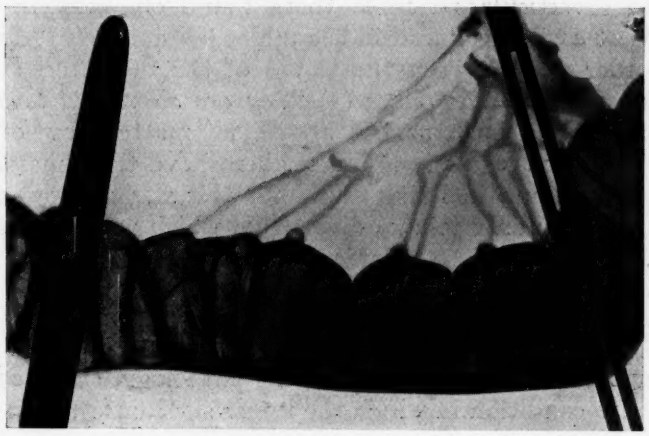

FIG. 133.—Artificially produced diverticula. Note each is related to an artery. Bismuth has been passed through the intestine prior to distension with oxygen. Forceps prevent the gas escaping.

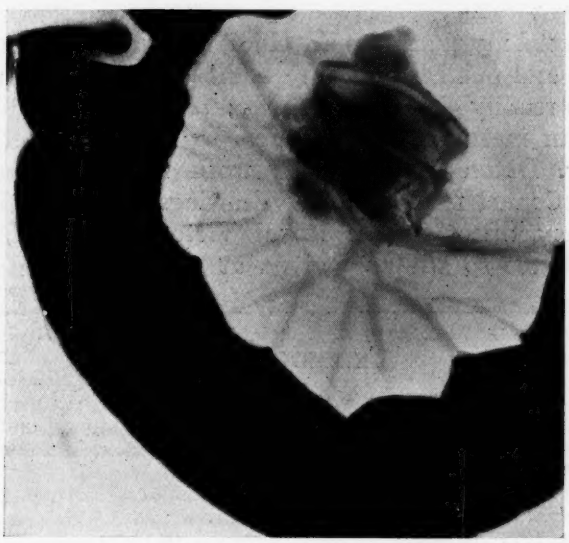

FIG. 134.—Diverticula are small, but their relationship to the mesenteric arteries is noticeable.

from this work the interesting fact resulted that equal distension in the jejunum, ileum, and colon produced diverticula in the jejunum only—which supports clinical facts entirely. Although diverticula are admittedly common in the colon, it is not held by many at the moment that they are the result of distension, which jejunal diverticula undoubtedly are.

The best pictures of these experimental diverticula were obtained by first filling the gut with a thin solution of bismuth and allowing it to run out before the gut was distended with oxygen. This method allows a clear delineation of the mucous membrane and shows that the diverticula produced are in reality herniæ of the mucous membrane through the muscle. *Fig.* 131 is an example of the artificial diverticula filled with bismuth. *Fig.* 132 shows the gut filled with oxygen, while *Figs.* 133 and 134 show in addition the fact that these artificial diverticula resemble those found clinically in almost every respect: (1) Most easily produced in the upper jejunum; (2) Always along the mesenteric border and between the leaves of the mesentery; (3) The sacs consist of mucous membrane only; (4) *They are all related to an artery.*

Gradual distension such as the above, no matter how often repeated, always gives the same results. Some workers have tried massive distension of the colon and rectum in living dogs, but this was without result, as one would expect, for it is only jejunal diverticula which are essentially the result of distension, as compared to colonic, which are the result of spasm.

CONCLUSIONS.

An attempt has been made to show the pathology, symptoms, and treatment of that rare condition in which the upper small intestine is studded with multiple false diverticula. The theories regarding the causation and production of such protrusions are considered, and a proposition is formulated, based upon the study of personal clinical material and personal experimental work.

I wish to record my thanks to my chief, Professor Fullerton, for permission to operate upon and publish the case described, which occurred in his wards; for his advice so readily given at all times; and for his kindly criticism which has ever been an incentive and stimulus to me to do better work. I am indebted to Dr. Dixon Boyd for his work in injecting the arteries; to Mr. W. A. C. McConnel for his outline drawings; and to Mr. Leman, of the Radiography Department in the Royal Victoria Hospital, Belfast, for his invaluable assistance so willingly given at all times.

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A CASE OF LIPOMA OF THE MESENTERY.

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Lipomata of the mesentery are classified by Crabtree[1] among the retroperitoneal fatty tumours. These are usually said to arise from the perirenal fat, but may also originate in fatty tissue in the renal capsule, in the intestinal mesentery, or in the omentum.

Von Wahlendorf reviewed 165 cases of retroperitoneal lipomata and found that 46 per cent of them were pure lipomata and 54 per cent were mixed tumours containing fibrous tissue. The latter are now considered to be sarcomatous in nature and not to be true lipomata.

True lipomata of the mesentery are very rare, and therefore the case recorded below may be of interest. Dannreuther[2] reports a case where the whole of the mesentery was infiltrated by a diffuse mass of lipomatous tissue in which the abdominal wall was not adipose but showed a remarkable atrophy of the abdominal muscles. Giampaoli[3] reported two cases of mesenteric lipomata in 1910 and Alton[4] another in 1921.

The central areas in a fatty tumour are very liable to necrosis from poor blood-supply. The occurrence of necrosis is said to give rise to fever, chills, and pain, but although the central portion of the tumour was necrotic in this case no febrile symptoms were apparent.

History.—A. H., a tube-worker, 22 years of age, was sent up to the Queen's Hospital, Birmingham, with a diagnosis of subacute appendicitis. He had been complaining of periodic attacks of pain in the stomach during the previous four months. These attacks, he said, were brought on by certain foods, such as fruit, currant-bread, or parsley sauce. The pain was described as a dull ache in the centre of the abdomen; it was not severe and disappeared after about half an hour. Three weeks before admission he had an attack of severe pain after eating bananas, this time accompanied by nausea which lasted an hour and a half. Two days before admission he received a slight blow in the abdomen from the handle of a gramophone he was winding. This was sufficient to bring on an attack of abdominal pain, which gradually subsided in the evening, but recurred the next day and became gradually more severe, until admission to the Queen's Hospital on the evening of Nov. 1, 1932.

On Examination.—The patient was found to be afebrile, but the abdominal muscles were rigid and he was rolling about in bed with pain, making examination difficult. A central abdominal distension was apparent with some visible peristalsis. Two enemata were given, the first producing a poor result and the second none at all. A diagnosis of acute small-intestine obstruction was made.

Operation.—An exploration was carried out through a right paramedian sub-umbilical incision. On opening the peritoneum an excess of free fluid was found and a tumour about the size of a large grape-fruit was encountered growing in between the layers of the mesentery of the small intestine. The gut surrounding it was stretched over its surface and closely applied to it anteriorly. The gut in relation to it had undergone a partial torsion and

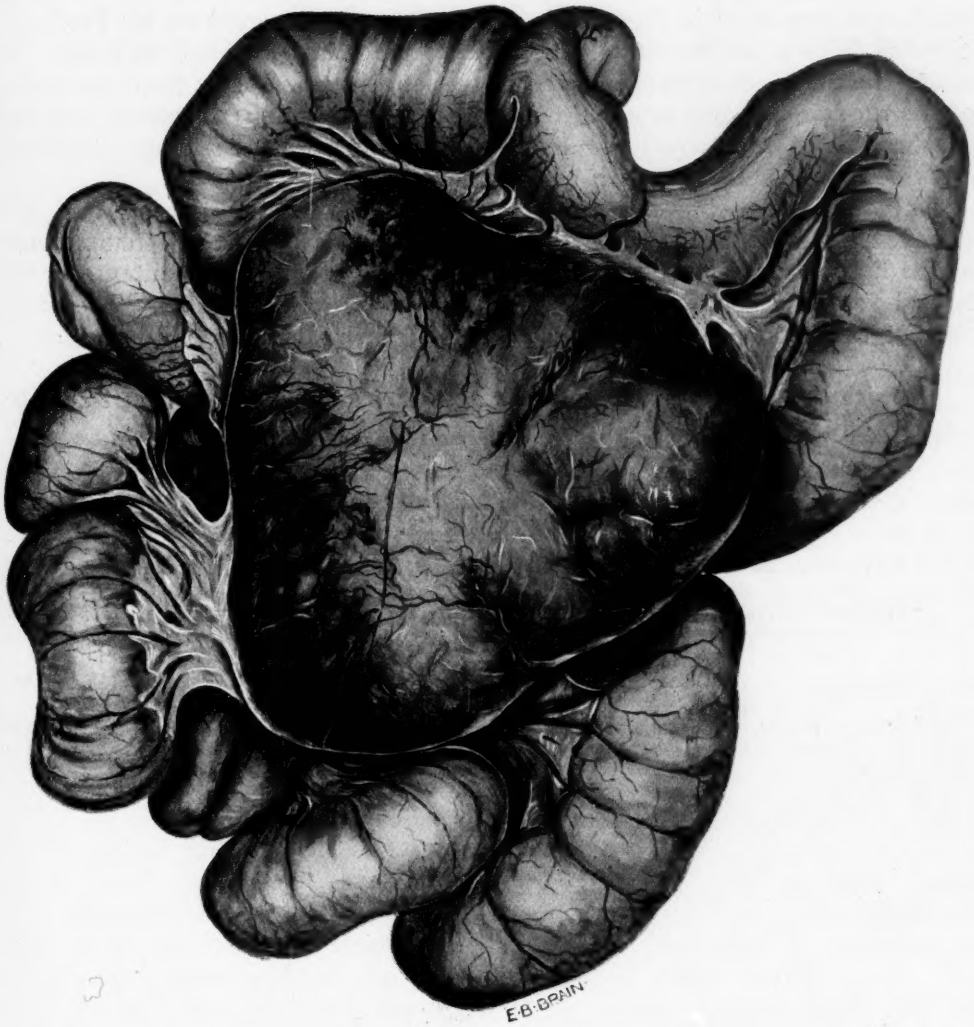

Fig. 135.—Showing the tumour and gut removed at operation.

was bluish in colour but viable. It was feared that attempts at enucleation might damage the blood-supply of an already weakened gut and so a wedge of mesentery containing the tumour, together with about 3½ ft. of gut in relation to it, was excised and an end-to-end anastomosis performed. To prevent undue tension on the line of junction a further side-to-side anastomosis was made between portions of gut above and below, and the abdomen thereafter closed without drainage.

SUBSEQUENT PROGRESS.—The patient made an uninterrupted recovery. His bowels acted without aid the day after operation, and when he began to take a light diet his bowel action was at first (for seven days) a little loose, but this became normal and regular at the end of that time. He left the hospital three weeks after admission, has returned to work, and continued in good health ever since.

PATHOLOGICAL REPORT.—A section from the surface of the tumour, which was preserved in its entirety, was taken and reported on by Professor Haswell Wilson as showing lipomatous tissue with areas of necrosis. To verify the diagnosis of lipoma a deeper section was taken later from the centre of the tumour, and was reported on on Dec. 17 as follows: "Sections from the deeper part of the growth showed the same appearances as those formerly described, viz., a lipoma with areas of necrosis".

The accompanying illustration (*Fig.* 135) shows the tumour and gut removed at operation. There are whitish areas around the circumference of the lipoma which were due to rupture and diffusion of the contents of the lacteal vessels and presented a remarkable picture at operation. The lower part of the tumour shows an area where necrosis had reached the surface.

I wish to thank Mr. Sankey, at whose direction I operated on this case, for permission to report it.

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OCCUPATIONAL ANEURYSM OF THE PALMAR ARTERIES.*

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A SURVEY of the literature of aneurysms of the palmar arteries yields records of 70 indubitable cases. Some references of older authors could not be traced, and some cases have been discarded on account of insufficiency of detail.

Like aneurysms elsewhere, those of the palmar arteries may be divided into false, of which there were 54 cases, and true aneurysms, of which 16 require consideration. The false aneurysms, usually referred to by the too inclusive and therefore somewhat misleading term 'traumatic aneurysm', resulted from punctured wounds of the hand, with a solitary exception when the aneurysm followed the subcutaneous rupture of an artery during reduction of a metacarpo-phalangeal dislocation of the thumb. This type of palmar aneurysm, following transfixion or laceration, does not come further within the purview of the present communication.

True aneurysm, a single, localized ectasia of one of the palmar arteries, is of relatively rare occurrence, and the majority of such cases seem to be traumatic in origin. A single case is recorded[18] in which the arterial affection, associated with subacute endocarditis, was considered to have followed the blocking of the vessel by an embolus. The likelihood of such an occurrence seems doubtful.

Where no obvious cause can be assigned, aneurysm of the palmar arteries has been termed 'spontaneous'. Only two cases of this variety are forthcoming, that of Griffiths,[3] an aneurysm of the ulnar artery in a young woman, and that of Reid,[15] where the aneurysm was not palmar but occurred on the back of the hand in a tobacco-dealer. Apart from these cases in which investigation failed to find adequate explanation for the aneurysm there are those which more nearly concern this paper, being traumatic in origin, though not dependent on an open wound. There are two varieties, both occupational, one acute and due to a single definite contusion, the other of more delayed and gradual development, the result of repeated minor injury.

Of the acute traumatic aneurysm of the palmar arteries, eight cases are recorded,[6, 10, 13, 14, 16, 17, 19] of which the following three may be abbreviated as types:—

W. Robertson[16] in 1897 described the case of a railway fireman at Perth, aged 18, who dealt the 'injector' of his engine a blow with the palm of his left hand, suffering considerable pain as a result. An aneurysm, probably of the ulnar artery in the hypothenar region, was noticed five months later.

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D. B. Robinson[17] places on record the case, in Kansas City, of a railway switchman who had also been a prizefighter. He struck the tap of an air-hose with the palm of the right hand and felt severe pain radiate to the fingers. One month later he came under treatment for an aneurysm of the deep palmar arch.

Regnault and Bourrut-Lacouture[14] published the case of a second master gunner in the French navy, aged 37 years. During firing practice the breech of his gun jammed and he dealt it several hard blows with the palm of his left hand in order to free it. He felt pain in the hypothenar region at the time, and three weeks later he was admitted to a naval hospital with an aneurysm of the hypothenar extremity of the superficial palmar arch.

The more strictly occupational palmar aneurysms, with which this paper is particularly concerned, are of that type which succeed prolonged and frequently recurring minor traumata, the trauma being, as a rule, incidental to the individual's work. This type best merits the term 'occupational' aneurysm, and of it only six cases are on record. Of these a short individual summary is of interest :—

Guattani.[4]—In 1772 there was published what appears to be the earliest fully recorded case of aneurysm of the ulnar artery in the palm. The side affected is not specified. More recent writers refer to the affected individual as 'coachman'[8] and attribute the aneurysm to the recurring trauma of handling reins and whip. Setting aside the fact that in 1772 throughout Continental Europe it was universal to ride postilion and not drive from the box, a study of Erichsen's classic translation from the original Latin of Guattani's paper[2] makes this man's real occupation clear. The passage in question runs as follows: "A groom, Johannes Perisius by name, a Roman, 45 years of age, of a sanguineous temperament, a great wine drinker and now the horse-keeper of the most excellent Marquis Angelo Gabrielli." Neither here nor elsewhere in the translation is there any mention of reins or whip, and the author does not draw attention to any chronic trauma to which the man was exposed. It appears reasonable, however, to suppose that a Roman groom in 1772 would be exposed to a considerable amount of arduous manual toil, and the case is therefore included here among the group of 'occupational' aneurysms.

Morestin.[12]—A carter, aged 43, whose work entailed the handling of many large and heavy boxes, developed a left ulnar aneurysm in the palm. The author concludes that the aneurysm is attributable to the man's occupation combined with a possible arterial degeneration due to an alcoholic disposition.

Duvernoy.[1]—A blacksmith, aged 50, developed an aneurysm of the thenar extremity of the right superficial palmar arch attributed to the repeated trauma of swinging a heavy hammer.

Mauclaire.[11]—A joiner acquired an aneurysm of the right superficial palmar arch as a result of the frequent use of a hand-plane.

Lejars.[7]—A book-carrier, aged 35, developed an aneurysm in the hypothenar region, caused apparently by the trauma of the edges and corners of the piles of books which he carried for a livelihood.

Marques and Marques.[9]—An aneurysm of the right deep palmar arch resulted from the recurring trauma of the implements used by an agricultural labourer.

To these records I am enabled to contribute the two following cases :—

Case 1.—A male, aged 38 years, attended the Surgical Out-patient Department of the Edinburgh Royal Infirmary towards the end of 1932. He was a healthy man in all respects save for the loss of his right leg during the war. His Wassermann blood-reaction was negative.

During ten and a half years he had earned a living as a wood-machinist. Most of his work was concerned with cutting six-inch lengths off long planks of wood. For this purpose he used a mechanical saw, and before cutting the wood he pushed the plank into position by a blow with the hypothenar region of his right hand. Furthermore, he had formed the habit, since the loss of his leg, of raising himself from the sitting position by pressing the palms of his hands against the edge or corners of the chair.

For some months past, while performing these movements, he had felt as if something deep in the hypothenar eminence slipped over an underlying bone. When this occurred pain was referred to the hypothenar eminence at a point about a finger-breadth distal to the pisiform bone. The pain did not radiate to the fingers, nor had he ever experienced paræsthesia referable to the ulnar-nerve distribution. There was no sign of ulnar-nerve weakness, but on careful palpation a small hard mass about the size of a pea could be felt to slip over the hook of the hamate (unciform) bone, and this movement reproduced the characteristic pain complained of.

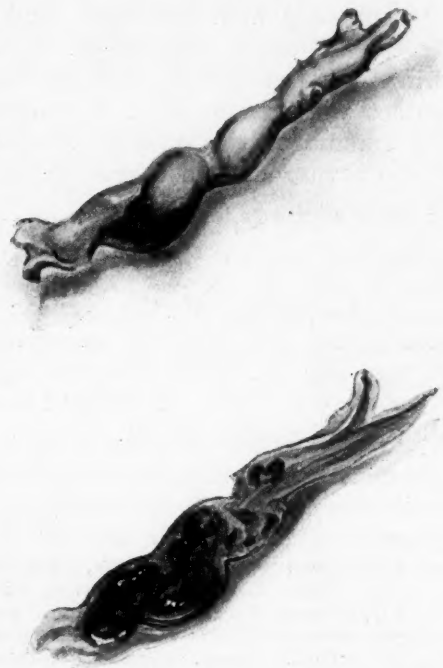

FIG. 136.—*Case* 1. Aneurysm of the ulnar artery in the palm.

At operation an incision was made in the line of the ulnar artery and nerve. These structures, where they entered the palm, were followed distally. Over the hook of the hamate bone the ulnar artery, which was more tortuous than normally, formed an aneurysmal swelling, which was filled with firm coagulum. The clot extended distally a short distance beyond the aneurysmal sac, and ceased abruptly where the artery divided into superficial and deep branches. The artery proximally and the superficial and deep branches distally were ligated. The intervening segment was then removed (*Fig.* 136).

Case 2.—I am indebted to Mr. D. M. Greig, Conservator of the Museum of the Royal College of Surgeons of Edinburgh, for permission to record an additional experience.

In 1888, while serving in India, Mr. Greig, as Orderly Officer in the Mian Mir cantonment of Lahore, was called to hospital to a private of the 33rd West Riding of Yorkshire Regiment who was bleeding from the right hand. The soldier was of more than seven years' service, and except for malaria had a clear medical history sheet; he denied syphilitic infection, and nothing to suggest syphilis could be found on general examination.

He had been admitted to hospital on account of a swelling in the right

thenar region. Diagnosed as an abscess, incision was followed by profuse hæmorrhage, which was controlled by packing. The hæmorrhage had recurred on more than one occasion and the tampon had been each time renewed. Mr. Greig ligated the superficial volar branch of the radial artery above and below an aneurysmal sac, and later the man returned to duty.

Investigation yielded a clear statement of the causal trauma. In firing his rifle he did not follow the drill-book instructions and brace the butt firmly against his body. He habitually helped to check the recoil by the strong grasp of his hands. He was always conscious of the recoil, not merely against his shoulder, but also against the base of his right thenar eminence on the 'grip' of the rifle. The military rifle in use in India at that time was the Martini-Henry. To replace each cartridge the breech was opened by depressing with the right hand a lever which lay close to and behind the trigger-guard. The action of sharply pushing down the lever was performed by the thumb, and the ball of the right thumb came abruptly in contact with the lever as it reached its limit of movement. Thus, recurring trauma to the superficial volar branch of the radial artery at the base of the thenar eminence was the main etiological factor in the production of an aneurysm.

SUMMARY.

1. Aneurysms of the palmar arteries are divided into: (*a*) False aneurysms; and (*b*) True aneurysms, which are frequently of occupational origin. The previously recorded cases of the latter are surveyed.

2. Two hitherto unpublished cases of true occupational aneurysms in the palm are placed on record.

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COCCYGEAL SINUS.

By R. L. NEWELL, Manchester.

I had some difficulty in choosing a title for this paper. Although coccygeal sinus or fistula is the name by which the condition is known in English literature, it is an inaccurate description because the only connection the sinus has with the coccyx is solely one of position. 'Congenital sinuses in the sacrococcygeal region' is perhaps a better title. In America Hodges[31] as long ago as 1880 manufactured the name of 'pilonidal sinus', or nest of hairs.

The condition has received very little attention by British surgeons. Goodsall and Miles[1] in their book on *Diseases of the Anus and Rectum* devote a whole chapter to a condition which they call 'sinus over the sacrum or coccyx', but they fail to recognize its true nature, and attribute its cause to injury. They recognize, however, that it has no connection with the coccyx or sacrum. Even as late as 1932 W. E. Miles[21] still regards it as due to a hæmatoma resulting from a fall, and advances the theory that right-handed people fall more heavily on the left side, accounting for the fact that these sinuses appear to originate on the left side of the median raphé.

Lockhart Mummery[2] drew attention to the congenital origin of the condition in 1921 and demonstrated that the sinuses were lined by epithelium, and later in 1929 published an excellent microphotograph in the *Proceedings of the Royal Society of Medicine*.[3] Towards the end of 1932 W. B. Gabriel[24] published his book *The Principles and Practice of Rectal Surgery*; in it he described the results of his treatment in 9 cases.

It is a condition, however, which has received considerably more attention in America. Harvey B. Stone[4] reports 61 collected cases. P. B. Cattell and L. W. Stoller[6] report 59 cases from the Lahey Clinic, and Frank Glenn[23] reports 120 cases. These figures give the impression that its occurrence is far commoner in America than it is in this country. Personally I am of the opinion that this is not the case, and that the lack of more frequent reference to it in our surgical literature is due to the failure on the part of the practitioner or surgeon to appreciate the pathological basis of the condition. Failure to understand the true nature of the sinus may lead to the mistaken diagnosis of fistula in ano, tuberculous sinus, or even a simple abscess or boil.

I do not doubt that there is hardly a surgeon in this country who has not seen or operated upon several of these cases, and possibly been disappointed with the result of his surgical intervention. P. B. Cattell and L. W. Stoller[6] have followed up 40 cases, and in 9 have found recurrences: 20 out of 50 of their cases had had previous operations.

SYMPTOMS.

The lesion seldom presents itself to the surgeon until the complication of septic infection has supervened. The following is a typical history.

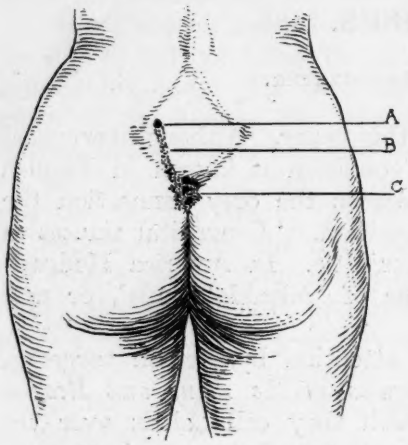

FIG. 137.—*Case* 1. A diagrammatic representation.

At an age of from 20 to 24 years a small abscess appears to one side of the middle line (*Fig.* 137, A). This is either incised or discharges spontaneously. After a few days the discharge ceases, but a small thickened nodule remains. The amount of pus is usually very small, and only rarely does the sinus discharge profusely. After an interval of a few days or weeks, this nodule again becomes painful, swells, and discharges pus. Sometimes the condition may remain quiescent for many months. This state of affairs goes on for an indefinite time until the condition is correctly diagnosed and the underlying cause removed.

On examination we find a small hard nodule to one side of the middle line, either temporarily healed over or showing a small opening with heaped up granulation tissue (*Fig.* 137, A). On palpation a firm hard core is felt leading downwards and towards the middle line (*Fig.* 137 B). In this position can be seen one or several tiny orifices situated accurately in the middle line (*Fig.* 137, C). These orifices may be situated quite close together or may be separated by a distance of as much as an inch. On pressure over the sinus at A and B (*Fig.* 137) pus may sometimes be seen to exude from the orifices in the median raphé.

If the sinus is injected at A (*Fig.* 137) with lipiodol, the solution will, after having filled up the whole of the track, be seen to exude from the orifices in the median raphé (*Fig.* 137, C). *Fig.* 138 shows an X-ray photograph after a lipiodol injection.

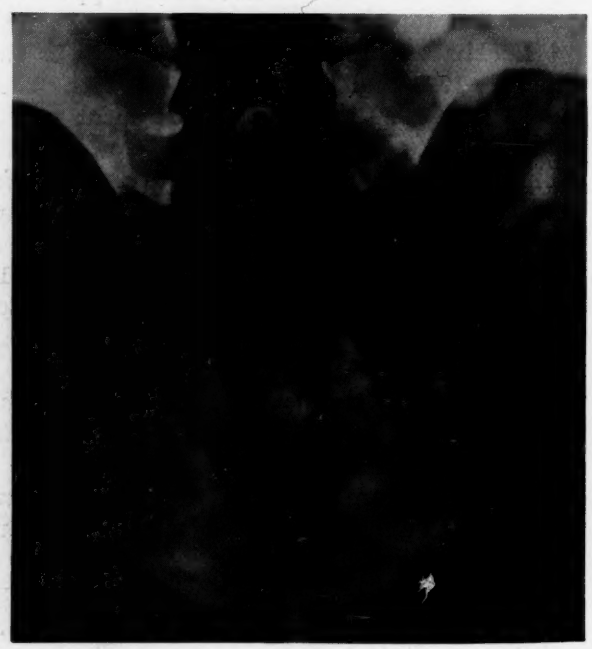

FIG. 138.—*Case* 1. X-ray photograph showing the sinus after a lipiodol injection. The upper narrow portion is the secondary sinus, the lower expanded portion the primary sinus in the median raphé.

The whole of the track is well outlined from A to the expanded cavity at C (*Fig.* 137). The narrow projection downwards below the expanded cavity is due to some of the lipiodol having escaped on to the surface of the skin.

HISTOLOGY.

A microscopic examination of the whole sinus track reveals the following appearance. The expanded cavity C (*Fig.* 137), which is the origin of the condition, is a cavity partially lined by true skin and may contain quite

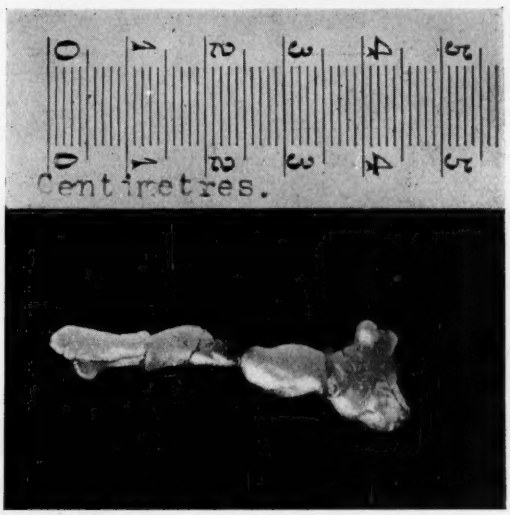

FIG. 139.—*Case* 1. Wax cast of the whole of the sinus. The thin portion to the left represents the secondary sinus. The expanded portion to the right, to which hairs can be seen adhering, represents the primary sinus.

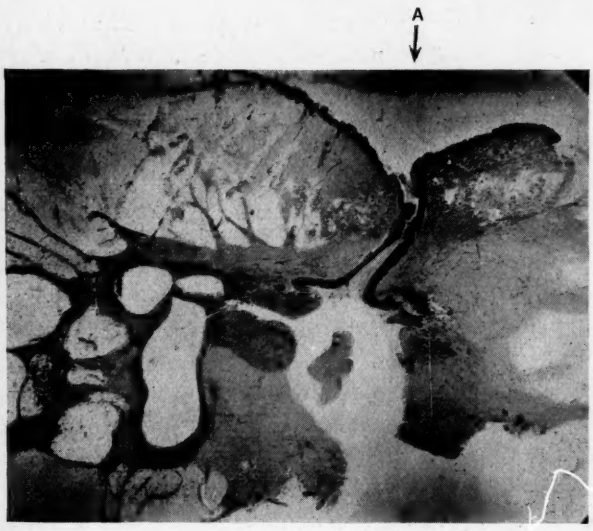

FIG. 140.—A horizontal section taken to show the connection of the primary sinus with the skin and deep tissue. A, Tiny orifice situated in the median raphé; B, Skin surface; C, Primary sinus lined by squamous epithelium; D, Epithelium expands into a cavity; E, Secondary sinuses lined by granulation tissue.

long hairs. Some hairs can still be seen adhering to the bottom of the wax cast seen in *Fig.* 139. This cavity communicates with the surface on the median raphé by orifices lined by true skin. These facts are well illustrated

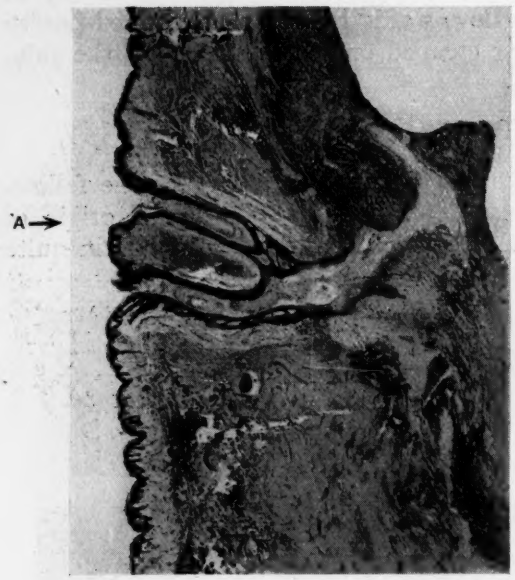

FIG. 141.—A section of specimen shown in *Fig.* 147 taken in the mesial plane. It shows two primary sinuses in the median raphé, one above the other. These two sinuses join and extend backwards towards the coccyx. The epithelium appears to be intact. The secondary sinus was caused by one of the other two primary sinuses present in this case. A, Two small primary sinuses in the median raphé; B, Secondary sinus.

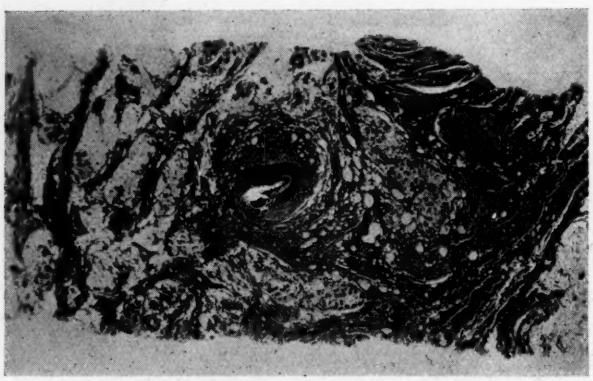

FIG. 142.—A vertical section in the coronal plane at right angles to *Fig.* 140.

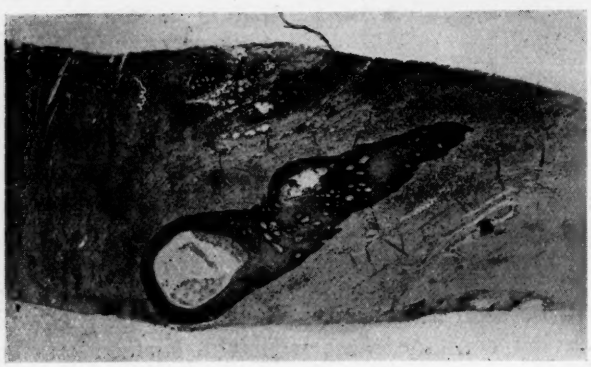

FIG. 143.—A vertical section nearer the coccyx than *Fig.* 142. The sinus is now expanding into a small cavity.

by the microphotographs (*Figs.* 140–144). The sinus B (*Fig.* 137) is purely a secondary sinus and is lined only by granulation tissue. This accounts for the fact that the pathologist, on examining the tissue removed, will often send back a report that it is septic granulation tissue.

FIG. 144. — High-power view of *Fig.* 143, showing a cavity containing hairs and epithelial débris and lined by epithelium.

ETIOLOGY.

The exact etiology of the condition has not yet been proved conclusively, but it probably results from change in embryonic development. Harvey B. Stone[4] gives to J. M. Warren[7] the credit of first describing the condition in 1867. Warren regarded a reversed hair follicle as being the cause of the trouble, and as the hair continues to grow "it pulls its hole in after it."

Wendelstadt[8] advances the following theory. The inferior end of the spinal cord is the last portion to lose its connection with the skin. As the sacrum closes together it pinches off this connection, and the skin becomes attached by fibrous tissue at this point to the posterior surface of the sacrum. As adult life approaches, fat and soft parts grow and lift the skin farther away from the bone, and the attached spot is drawn down into a dimple or sinus. P. B. Cattell and L. W. Stoller[6] give Oehlecker's[9] views without comment. He believes that these sinuses are caused by the traction of the caudal ligament on the median raphé. Hermann and Tourneaux[22] regarded the condition as due to a vestigial remnant of the spinal cord. They state that after the separation of the spinal cord from the superficial tissues a process of epithelial tubules persists for a time at the point of previous connection of the neural axis with the skin, forming a vestigial remnant of that connection.

Harvey B. Stone,[5] in a more recent paper, suggests as a possible cause that the sinuses are analogous to the preen glands found in a great many species of birds. The preen gland lies embedded in fat over the last caudal vertebra. It consists of numerous straight tubules lined by polyhedral cells. The tubules converge upon a collecting chamber or cavity, which in turn empties into an epithelial-lined duct opening on to the skin of the back. Their function is either that of oiling feathers or it is sexual.

Ripley[10] and Moise[11] both regard the condition as analogous to spina bifida, and have recorded two cases of staphylococcic meningitis resulting from a congenital sacral sinus. In both their cases there was, however, a direct communication with the spinal meninges and a maldevelopment of the vertebral bodies. Bland-Sutton[12] considers that the condition is due to faulty coalescence of the cutaneous covering of the back, and mentions as analogous the interdigital pouch of the sheep, which often gives trouble if

the orifice becomes occluded. He says that they are sinuses frequently associated with lumbo-sacral spina bifida. This statement, however, is not corroborated by an analysis of the published cases.

It would appear, then, that we have to consider two main theories :—

1. That the condition is a true sequestration dermoid, due to faulty coalescence of the middle line.

2. That the condition arises from that remnant of the spinal canal known as the coccygeal vestige.

I do not think the condition is entirely analogous to a true sequestration dermoid. Coccygeal fistulæ are comparatively frequent, and are in a constant position, whereas dermoid fistulæ arising in the middle line are very rare in other parts of the body. I therefore consider that the area in which they are found has a definite influence on their etiology.

As regards the theory that they arise from the coccygeal vestige, we have the work of Hermann and Tourneaux[22] to support this hypothesis. They showed that in the fœtus at a definite stage there is a connection of the coccygeal vestige with the skin. On the other hand, recent work by Kunitomo,[28] who had available for his research the unrivalled collection of embryos at the Carnegie Institute, has failed to confirm this statement. Kunitomo states, "The caudal end of the coccygeal medullary vestige appears to adhere to the epidermis, but in reality it does not."

What, however, is very significant in his work is that he found the various stages in the reduction of the tail bud, as shown on the skin, do not present the same appearance in every embryo, but, on section, evidence of its reduction and disappearance are invariably found dorsal to the caudal end of the vertebral column and in the median line of the embryo.

A coccygeal fistula is lined by tissue indistinguishable from true skin. If it arose from the coccygeal vestige, the cells lining it would have become so differentiated as to be quite unlike true skin. In microscopic sections of the condition there is absolutely no sign of any other tissue than that normally contained in the skin.

I venture to suggest that the following is the true origin of the condition. The sinus is a dermoid fistula, but differing from a true sequestration dermoid in that it is caused by traction of the underlying tissue upon the median raphé. This traction is probably caused by the retrogression of the tail bud. It therefore could be described as a traction 'dermoid'. It may be suggested that the following is the probable sequence of events. The epithelial-lined fistula is present at birth. It would appear to remain a considerable time without giving rise to symptoms. However, owing to the growth of the body and consequent enlargement of the buttocks, the secretion of this epithelium is unable to escape by the orifices in the middle line. These orifices are in a position which is extremely liable to sepsis, being situated at the bottom of a fairly deep cleft. The epithelial fistula becomes infected, and the formation of an abscess follows. This abscess disintegrates a portion of the epithelial lining and a sinus track is formed which tends to extend along the line of least resistance, which is upwards and outwards. This secondary sinus eventually opens on to the surface to one side of the middle line.

CASE REPORTS.

The following cases have been operated upon by me by the method I am about to describe. In all cases the condition appears to have been completely cured.

Case 1.—M. C., female, aged 26. The sinus had been discharging for twelve months. There were three primary sinuses in the middle line and one secondary sinus opening on the left of the middle line. Operation on Feb. 5, 1932. The wound became septic and healed by granulation tissue.

Case 2.—C. J., female, aged 30. The sinus had been discharging for two years. She gave a history of one previous operation. There were two primary sinuses in the middle line and one secondary sinus opening on the left of the middle line. Operation on Feb. 10, 1931. Wound healed by first intention.

Case 3.—G. A., female, aged 37. The sinus had been discharging for two years. She gave a history of one previous operation. There were three primary sinuses in the middle line and one secondary sinus opening on the right of the middle line. Operation on July 26, 1932. The wound became septic and healed by granulation tissue.

Case 4.—M. P., female, aged 20. The sinus had been discharging for two and a half years. She gave a history of one previous operation. There was one primary sinus only in the middle line. Operation on Sept. 29, 1932. The wound healed by first intention.

Case 5.—E. H., female, aged 25. The sinus had been discharging for ten years. She gave a history of three previous operations. There were two primary sinuses in the middle line and one secondary sinus opening on the left of the middle line. Operation on May 31, 1932. The wound became septic and healed by granulation tissue.

Case 6.—H. P., female, aged 25. The sinus had been discharging for four years. She gave a history of one previous operation. There were two primary sinuses in the middle line and one secondary sinus on the left of the middle line. Operation on Nov. 4, 1931. The wound became septic and healed by granulation tissue.

Case 7.—F. H., female, aged 30. The sinus had been discharging for six months. There was one primary sinus in the middle line and one secondary sinus to the left of the middle line. Operation on Feb. 8, 1933. The wound became septic and healed by granulation tissue.

Case 8.—E. W., female, aged 32. The sinus had been discharging for twelve years. She gave a history of two previous operations. There were four primary sinuses in the middle line and one secondary sinus on the left of the middle line. Operation on Feb. 10, 1933. The wound became septic and healed by granulation tissue.

Case 9.—K. B., female, aged 23. The sinus had been discharging for two and a half years. She gave a history of several incisions. There were four primary sinuses in the middle line and one secondary sinus on the left of the middle line. Operation on Feb. 14, 1933. The wound became septic and healed by granulation tissue.

Case 10.—M. B., female, aged 23. The sinus had been discharging for one year. She gave a history of several incisions. There were two primary sinuses in the middle line and one secondary sinus on the left of the middle line. Operation on Jan. 31, 1928. The wound became septic and healed by granulation tissue.

Case 11.—A. S., female, aged 20. The sinus had been discharging for two years. She gave a history of two previous incisions. There was one primary sinus in the middle line and evidence of a healed secondary sinus to the left of the middle line. Operation on March 17, 1933. The wound became septic, and is healing by granulation tissue at the time of writing.

All these cases occurred in women. Frank Glenn's figures show, however, that it occurs more frequently in men. There were 23 females and 97 males in his 120 collected cases. The earliest onset of symptoms was at the age of 15 years, and the latest onset at the age of 35 years.

All these cases were septic at the time of operation, and primary healing was obtained in only two of the series. The remainder healed by granulation tissue. The time taken for complete healing varied from three to ten weeks. In no case have the symptoms recurred.

TREATMENT.

Although successful treatment has been reported with non-surgical methods by Maillard[13] and Crookall,[14] it would appear that the only satisfactory way of curing the condition permanently is by surgical excision. A complete radical excision which removes the whole of the area involved is absolutely necessary, otherwise recurrence is inevitable. Failure to undertake a preliminary investigation of these cases with a view to obtaining a true diagnosis and an accurate delimitation of the sinus results in many of the patients having to undergo several operations before obtaining a cure. The following method has been found to give very satisfactory results :—

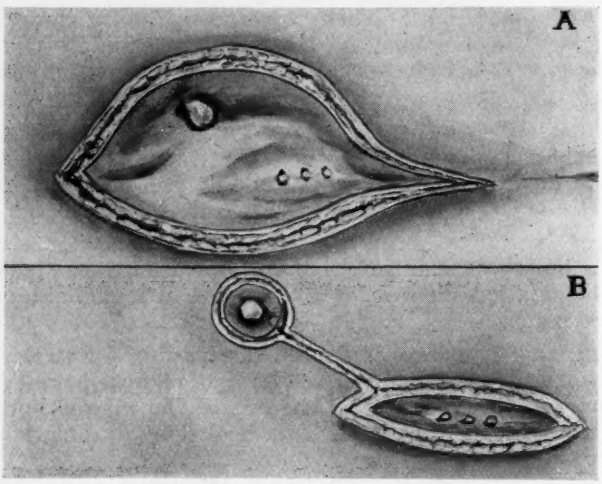

FIG. 145.—A, The incision used when the secondary sinus opening is near the openings of the primary sinuses; B, The incision used when the secondary sinus opening is situated some distance away from the openings of the primary sinuses.

The secondary sinus opening (*Fig.* 137, A) is injected with lipiodol. A blunt needle of fairly wide bore is used so as to fit tightly in the opening. It will be noticed on injecting the fluid that the lipiodol will begin exuding from the tiny orifices in the middle line (*Fig.* 137, C). Excess of lipiodol must be carefully wiped away from the cleft between the buttocks. An X-ray photograph is now taken without disturbing the patient. This photograph gives a fairly accurate idea of the extent of the condition (*see Fig.* 138).

Immediately before the operation, when the patient is under the anæsthetic, the opening of the secondary sinus (*Fig.* 137, A) is injected with melted paraffin wax. Coloured wax can be employed, but there is no advantage in this, as the wax is only used to make the whole of the sinus palpable and to serve as an indication if the sinus is cut across during the operation. A circular incision is now made around the orifice of the secondary sinus, and a straight incision directly over the underlying secondary sinus is made downwards towards the middle line (*Fig.* 145B). That portion of the median

raphé which is affected is now excised by an elliptical incision. If the secondary sinus opening lies near the middle line, then the incision must include both primary and secondary openings (*Fig.* 145A). The incision in *Fig.* 145B will, however, facilitate the closure of the wound. The skin-flaps are dissected well back, taking care to keep fairly superficial (*Fig.* 146). The whole of the sinus track is now removed (*Fig.* 147), beginning at the upper portion. There is no need to make a wide excision of the track from A to B (*Fig.* 137), as the track in this situation is lined only by granulation tissue and has no epithelium lining its walls. The condition, however, is different when the middle line is approached. Here we are dealing with the primary sinus, which has an epithelial lining, and it is advisable to excise not only the sinus but also the skin of the median raphé and a generous portion of tissue around the sinus. Otherwise it is possible that any pits or depressions of the epithelium lining may be cut across and left behind.

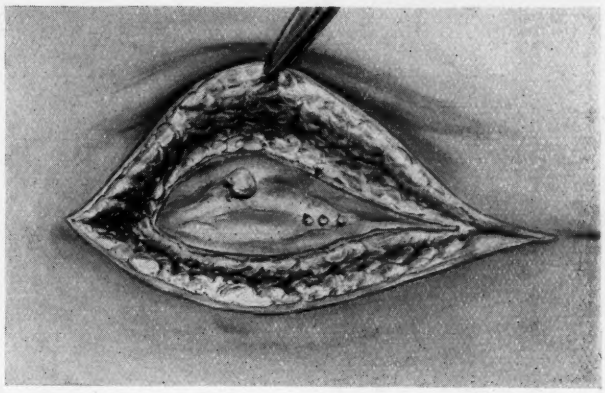

FIG. 146.—The skin edge is undermined.

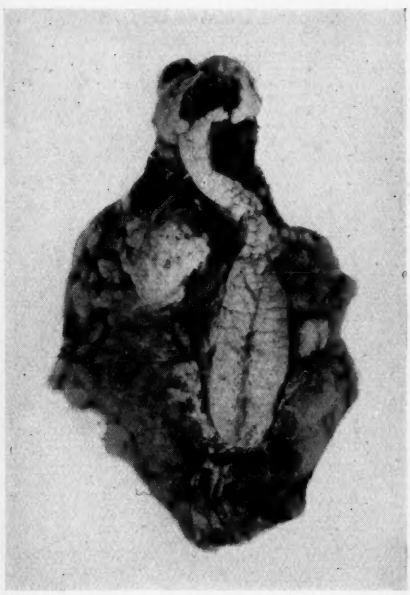

FIG. 147.—*Case* 8. Photograph of the sinus removed. The lower portion of skin shows four small openings in the median raphé. The narrow portion of skin passing upwards and to the left is overlying the secondary sinus. The opening of the secondary sinus can be seen at the top of the specimen.

This dissection will, of course, leave a fairly large cavity extending forwards to the coccyx, and an attempt must be made to obliterate this cavity as far as possible by undermining the fat and approximating it with catgut sutures. The skin is then sutured without drainage. An application of new skin helps to prevent contamination of the wound. If the wound becomes septic, as is not improbable, then the sutures must be removed and the wound allowed to heal by granulation tissue. Irrigation with a eusol solution should be performed twice daily, and later light packing with an allantoin solution will be found very efficacious in stimulating granulation tissue formation. Every effort should be made to obtain primary healing by swabbing the cavity with B.I.P.P., by undermining the fat, and by obliteration of the cavity with as little tension as possible by means

of catgut sutures through the fat. If primary healing is not obtained, it may be as long as two months before healing by granulation tissue takes place.

CONCLUSIONS.

1. A coccygeal sinus must be regarded as due to a defect in embryonic development. It probably is the result of traction on the skin caused by retrogression of the tail bud.

2. The treatment consists in the removal of the whole of the sinus, together with that portion of the median raphé which contains the origin of the condition.

3. The extent of the sinus may be difficult to recognize without a lipiodol injection, followed by an X-ray examination.

4. The dissection is rendered easier by an injection of paraffin wax immediately prior to operation.

I wish to tender my thanks to Mr. John Morley and Professor J. S. B. Stopford for their helpful advice in the preparation of this paper, and to Dr. G. E. Loveday, and Dr. E. R. A. Cooper for the preparation of the microscopic sections.

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ASSOCIATED FACIAL AND INTRACRANIAL HÆMANGIOMATA.*

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The different forms to which anomalous development of angioblastic tissue may give rise in connection with the central nervous system and its envelopes are gradually being recognized. The presence of such lesions, though rare, can in some instances be suspected, but the question—at times a difficult one—of their most satisfactory treatment is still under consideration.

The following case exemplifies an extensive and somewhat rare distribution of hæmangiomatous tissue in connection with the fore-brain and its coverings.

CASE REPORT.

On Dec. 3, 1931, a well-built boy, aged 8, was admitted to the Surgical Unit on the recommendation of Dr. Ivor Davies, who had seen him in his out-patient department. The child's mother stated that he had been subject to fits for about seven years, and that the fits occurred at intervals of approximately three weeks. Each fit was characterized by loss of consciousness, lasted for about two or three minutes, and was followed by " paralysis of his left side ", which sometimes remained as long as three days but then passed off.

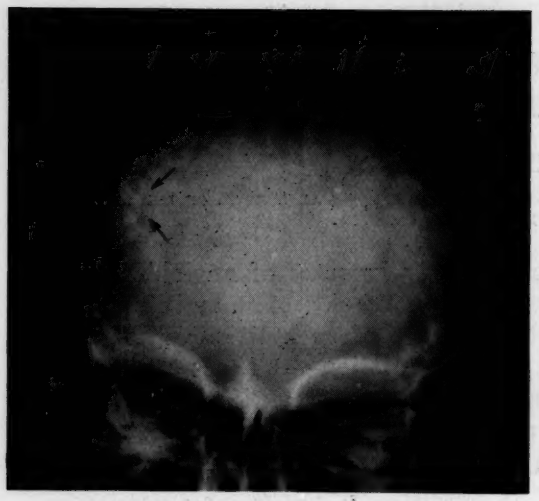
On Examination.—Examination revealed an extensive 'port-wine' stain over the right side of the face and forehead (*Fig.* 148), brisk knee-jerks, the left a little brisker and more pronounced than the right, and flexor plantar reflexes. Nothing else abnormal was noted.

A report on the fundi was made by Dr. Leighton Davies, as follows: " There is no optic neuritis in either eye, but the left disc appears a little abnormal. It is almost impossible to get more than a fleeting glimpse, but apparently the centre of the disc is very pale. I think this is probably due to an abnormally deep and large physiological cup. ? one small hæmorrhage in left retina. No engorgement of vessels."

Fig. 148.—Boy, aged 8, with fronto-parietal extra- and intradural plexiform angiomata on the same side as an extensive capillary nævus, visible in the photograph.

* From the Surgical Unit, The Welsh National School of Medicine, The Royal Infirmary, Cardiff.

While the patient was in hospital the fits became more frequent; three occurred over one week-end. A characteristic fit is described as follows: "There was no cry, the arms and legs were thrown about, and all four limbs twitched violently. Breathing was laboured and rapid." After certain, but not all, fits there was a left hemiparesis.



FIGS. 149, 150.—Antero-posterior and right lateral X-ray appearances of patient shown in *Fig.* 148. An opacity can be seen in the right frontal region.

The skull was X-rayed by Dr. Owen Rhys, and plates showed an opacity in the right fronto-parietal region (*Figs.* 149, 150).

DIAGNOSIS.—Owing to the increasing frequency of the fits followed by transient attacks of left hemiplegia, and the X-ray evidence of an intracranial opacity, it was decided to carry out an exploratory osteoplastic craniotomy over the right frontal lobe. A tentative diagnosis of a partly calcified hæmangioma was made, because of the attacks of convulsions followed by fleeting hemiplegia and the presence of the facial nævus.

FIRST OPERATION.—On Jan. 25, 1932, under intrapharyngeal chloroform and ether anæsthesia, an osteoplastic flap was cut with the skull plough. The flap was designed to expose the outer aspect of the right frontal lobe and to have its base beneath the temporal muscle. The bone-flap was turned down and the exposed dura mater was seen to be tense and not pulsating. The middle meningeal vessels passed upwards and backwards across it, and in the middle of the field an intricate anastomosis of fine branches lay across the main meningeal vessels, forming a plexiform angioma about the size of a large almond (*Fig.* 151).

The meningeal vessels were ligated with fine catgut as near their origin as possible. The dura was opened well away from the edges of the blood-vessel tumour, some outlying branches of the meningeal vessels being divided between silver clips. The frontal lobe bulged slightly through the wound as the dura was divided, and the whole surface of the exposed cortex was found to be beset with fine anastomosing vessels. The dura mater was anchored to the brain by a leash of vessels which passed from the deep surface of the extradural hæmangioma to the face of the similar but more extensive cortical tumour. Silver clips were placed on the vascular leash, which was afterwards divided between them so that the dural flap could then be thrown backwards on its base (*Fig.* 152). The brain was now seen to be pulsating normally. A small subtemporal bone defect was produced for decompression at the base of the bone-flap, the dura mater laid back over the cortex, and the wound closed after replacing

the bone-flap. The epicranial aponeurosis was sutured separately with fine silk and the skin with interrupted silkworm-gut sutures. *Fig.* 153 shows the X-ray appearances after operation.

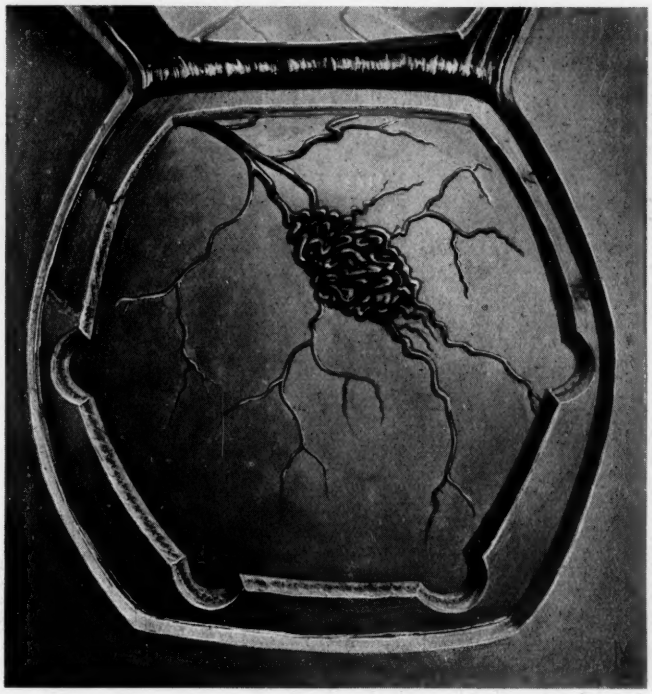


FIG. 151.—Appearance of extradural plexiform hæmangioma at operation.

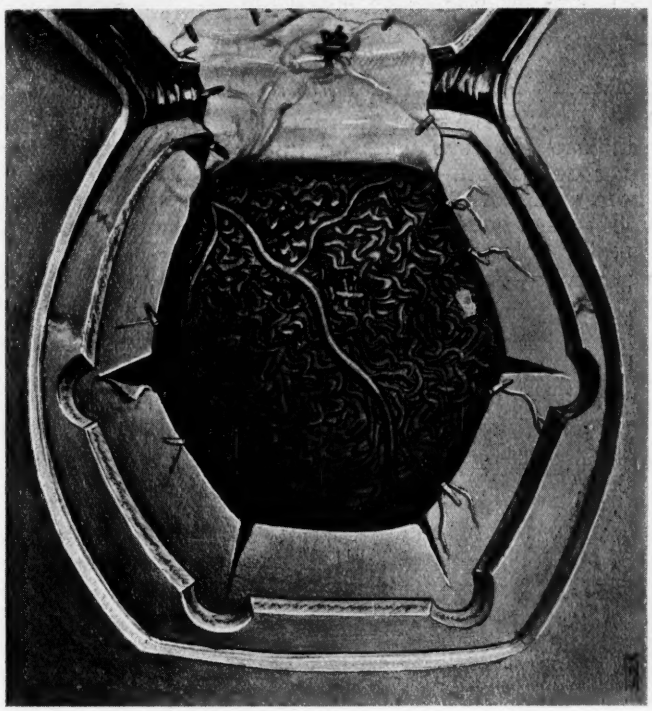


FIG. 152.—Appearance of intradural plexiform hæmangioma as revealed on opening the dura mater.

SECOND OPERATION.—Convalescence was uneventful except that two fits occurred, one three days and the other twenty-two days after the operation. Neither of these was severe and neither was followed by hemiplegia. It was thought advisable, however, to attempt further to interrupt the blood-flow through the lesion, and

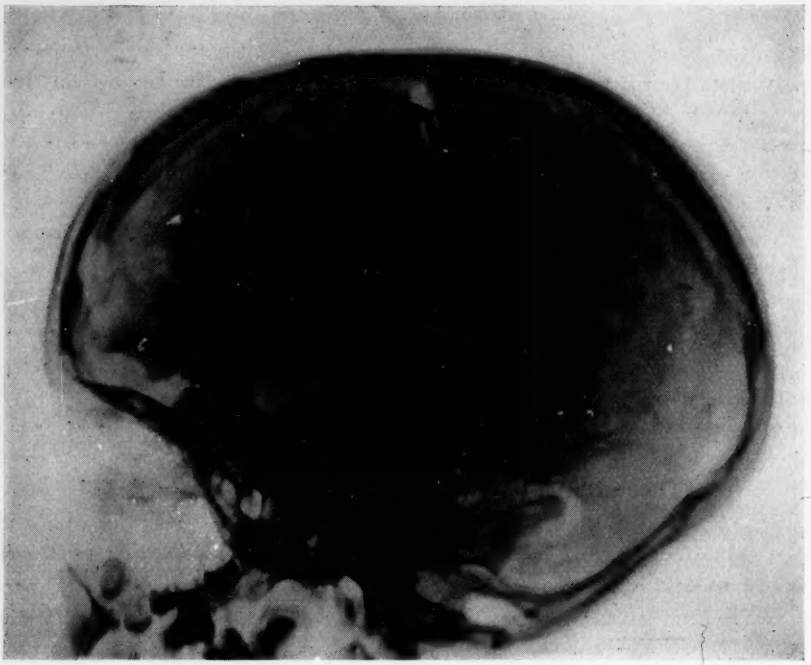


FIG. 153.—X-ray appearances after operation. The position of the bone-flap, with the subtemporal decompression, is shown. The small dark shadows are made by silver clips occluding vessels.

accordingly after a few weeks in a convalescent home, the patient was re-admitted, and on April 1, 1932, the right internal carotid artery was occluded with two catgut ligatures applied just beyond its origin from the sinus caroticus.

PRESENT CONDITION.—The patient was discharged from hospital on April 13, and has been seen from time to time. There have been one or two fits of a mild character, but these have not been followed by hemiplegia and the boy has returned to school and is able to lead a normal life.

Commentary.—A case is here recorded of extensive extra- and intradural plexiform arteriovenous anastomoses connected with each other, and associated with a facial capillary hæmangioma (nævus) on the same side. The association between the facial and intracranial hæmangiomata is a well-recognized one, instances having been recorded in 1902 by Cassirer,[1] and since then by Harvey Cushing,[2] Hebold,[3] E. Sachs,[4] Dimitri,[5] Marque,[6] and others; but the very extensive character of the intracranial lesions in this case is unusual, as also is the fact that the intracranial lesion was apparently a serpentine arterial angioma, whereas the intracranial angiomata associated with facial nævi apparently are usually of the venous variety (Cushing and Bailey,[7] 1928).

The clinical picture—namely, convulsions immediately followed by evanescent attacks of hemiplegia, and with little or no general progression of the

signs and symptoms—is characteristic of these intracranial blood-vessel lesions. Calcification, such as is shown in the X-ray plates of this case (*see Figs.* 149, 150), may or may not be present.

TREATMENT.

Two questions arise in connection with the treatment of these cases: (1) Is any treatment indicated? (2) If so, what should be its nature?

The answer to the first question would appear to depend upon certain subsidiary ones—namely: What is the outlook for the untreated patient, can this outlook be helpfully modified by treatment, and, finally, will such treatment be palliative as far as symptoms are concerned?

Life-history.—W. E. Dandy[8] (1928) found that of 22 cases collected from reports, 9 had died of intracranial hæmorrhage, while 2 from his own group of 8 cases had had hæmorrhages from which they had recovered. Such patients, therefore, are faced with an ever-present danger of intracranial hæmorrhage, which if it takes place, is usually fatal. Ernest Sachs[9] (1931) and others have recorded cases which have been successfully operated upon and relieved from symptoms. Sachs' case was a boy aged 10, with a facial port-wine stain, normal fundi, and attacks of convulsions. He was operated upon in February, 1915, an osteoplastic exploration being performed and occlusion of some of the vessels associated with the lesion, which from the description appears to have been somewhat similar to the one here reported. The patient was much improved, his fits diminishing and finally ceasing within a few months of leaving hospital. He then became and has since remained perfectly well. There would thus appear to be sound reason combined with precedent for operating upon such cases.*

The second question—What should be the nature of the treatment?—requires careful consideration. The temptation to do too much is great. Attempts to extirpate the vascular mass are to be deprecated. There may appear to be indications to apply many ligatures to the multitude of vessels comprising the lesion, but if such a procedure is followed, there is an immediate danger of hæmorrhage, which, if it occurs, may be difficult to control owing to the defective nature of the walls of the vessels; or if this danger is averted, there is the subsequent one of a persisting monoplegia or hemiplegia.

It is probably because of these considerations that Cushing and Bailey[7] give as their summing-up to the question of treatment of the cerebral venous angiomata: "The lesions, in short, when accidentally exposed by the surgeon had better be left alone." In the case of certain of the arterial angiomata, however, they appear to favour radiotherapy, decompression, and carotid ligations. Sachs' case and the one here recorded would appear to justify a certain degree of guarded surgical interference, efforts being made to bring about partial vascular occlusion by means such as have here been described.

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* E. Sachs has recently reported (*Southern Medical Journal*, 1932, Oct., xxv, 1013) a method of treating these cases by electrosurgery; the vessels are gently stroked by an electrode conveying a weak coagulating current. He points out that coagulating large vessels is tedious and must be done slowly, and that if the current used is too strong, the vessel being stroked is perforated and a large hæmorrhage occurs; but he has now been able to apply this method successfully in several cases.

THE NATURE OF THE LESION.

Such lesions as that presented by this case do not appear to represent true blastomatous tumours, but rather maldevelopments of the original vascular bed from which arteries, veins, and capillaries normally develop.

Dandy[8] quotes from Virchow's *Krankhaften Geschwültse* to show that the great German pathologist was aware of the true nature of these lesions: "There remains that form of angioma in which the vascular enlargement is outspoken and in which the character of the tumour is more in the background . . . one cannot make a true line of demarcation between them and angiomata. Their difference lies principally in the fact that the process is diffuse and that dilatation of both arteries and veins is present." Regarding them as arterio-venous aneurysms, Dandy believes that the varying appearances which they present depend upon the arterial and venous channels concerned in their formation and the nature of the intervening vascular bed. Cushing and Bailey,[7] however, while classifying the blood-vessel tumours of the brain in two groups—namely, the vascular malformations and the hæmangioblastomas—subdivide the former, which are most commonly found in the cerebral hemispheres, into teleangiectasis, angioma venosum, and angioma arteriale; and the latter, which are almost exclusively limited to the cerebellum, into cystic and solid varieties. The separation into two main groups is indisputable and very desirable, but the subdivision of the malformations would appear to be less so, since intermediate types occur between the three sub-varieties, and Dandy's explanation of the cause of these modifications appears to be a reasonable one and to reduce them to a common basis.

CONCLUSION.

It is possible at times to make a pre-operative diagnosis of intracranial blood-vessel tumour of the malformation variety. The symptoms produced by such a lesion may be alleviated, and the danger of fatal intracranial hæmorrhage averted, by osteoplastic exploratory craniotomy with occlusion of the principal source of blood-supply to the lesion.

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POUCHES OF THE PHARYNX AND ŒSOPHAGUS WITH SPECIAL REFERENCE TO THE EMBRYOLOGICAL AND MORPHOLOGICAL ASPECTS.

(*The Arris and Gale Lecture delivered before the Royal College of Surgeons, Feb.* 17, 1933.)

By R. W. RAVEN, London.

Pouches of the pharynx and œsophagus have been subject to much speculation for nearly two hundred years. Contributions have been made by many orders of our profession—surgeons, physicians, morphologists, and embryologists. In spite of this there is probably no other subject in surgery in which so many inaccurate observations have been made or so many erroneous theories perpetrated. At the present day the literature is full of errors, and these are present even in the latest standard works of surgery. In this paper I wish to consider the subject especially from the embryological and morphological aspects. Certain facts already known will be emphasized, doubt will be cast on some views which are held, and several new varieties of pouches will be described.

CLASSIFICATION.

Pouches of the pharynx and œsophagus may be classified as follows :—

Pouches of the Pharynx.—

1. *Congenital :*
   - *a.* Lateral pouch in connection with second embryonic endodermal pouch.
   - *b.* Lateral pouch derived from third embryonic endodermal pouch.
   - *c.* Lateral pouch derived from fourth embryonic endodermal pouch.
2. *Acquired :*
   - *a.* Posterior pulsion.
   - *b.* Anterior pulsion.

Pouches of the Œsophagus.—

1. *Congenital :*
   - *a.* Associated with œsophago-tracheal fistula.
   - *b.* Associated with imperfect separation of œsophagus and trachea.
   - *c.* Associated with localized imperfect development of muscle coats.
   - *d.* Associated with multiple pouches of the colon.
   - *e.* Single pouch in the posterior wall.
2. *Acquired :*
   - *a.* Tuberculous pouches.
   - *b.* Secondary to œsophageal stricture.
   - *c.* Secondary to œsophageal ulceration.

POUCHES OF THE PHARYNX.

CONGENITAL POUCHES.

Development of the Pharynx. — In all Craniota bilateral symmetrical pouches are formed from the anterior region of the foregut. At the same time corresponding invaginations form in the ectoderm. As a result of outward growth, the pouches press aside the lateral mesoderm of the head and come into apposition with the corresponding ectodermal invaginations (*Fig.* 154). The temporary fusion of endoderm with ectoderm forms the epithelial closing membrane.

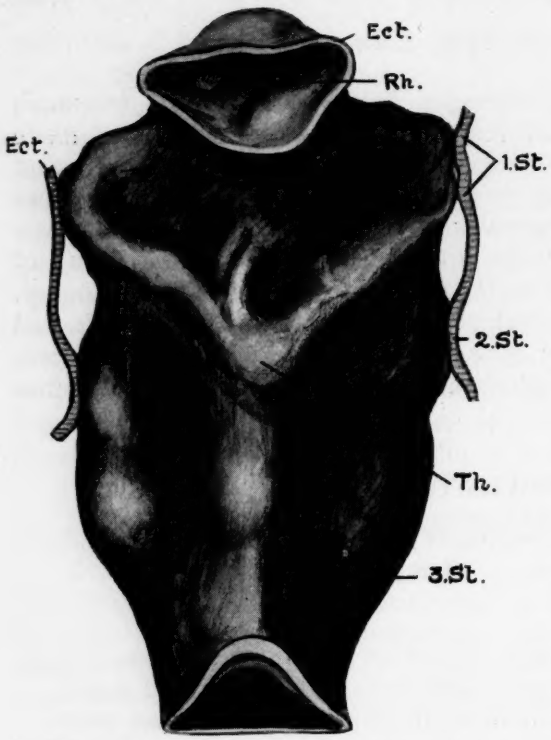

FIG. 154.—Pharynx of embryo about 3 mm. Ect., Ectoderm; Rh., Pharyngeal membrane; Th., Thyroid; St., 1st, 2nd, 3rd pharyngeal pouches. (*After Keibel and Mall*—"*Human Embryology*".)

In animals with branchial respiration, clefts are formed by perforation of the epithelial closing membrane; the number of branchial clefts varies from five to nine in different forms. The closing membrane in the human embryo and in the majority of mammals remains imperforate, and open clefts do not normally occur.

As growth proceeds, dorsal and ventral angles are formed in each pouch with the exception of the first, and simultaneously the epithelial closing membrane elongates. The connection of each pouch with the pharynx becomes constricted owing to the disparity of growth between the lateral part and the medial part; this constriction forms the endodermal ductus pharyngo-brachialis.

The structure of the early pharynx is very transitory. The entire region is in a state of continuous growth. As a result of the disproportionate size and growth of the outer pharyngeal arches, the precervical sinus appears on the surface at the early stage of 5 mm.

I wish to call attention to the broad strip of thickened ectoderm running from before backwards, along the upper ends of the arches and grooves (*Fig.* 155). This is the placodal area. In certain parts the cells of this area are continuous with the cellular masses of the nerve rudiments of the pharyngeal arches; two of these parts are indicated in *Fig.* 155 by dotted lines and are the placodal areas of the ninth and tenth cranial nerves. The ninth nerve placode is situated at the upper end of the third arch, some distance down the side of the second groove.

The Ultimate Fate of the Ninth Nerve Placodal Area and its Connections.—The importance of the ninth nerve placodal area in the interpretation of certain vestigial structures in the neck has been overlooked.

The area of the precervical sinus deepens markedly owing to the rapid growth of its boundaries. There is also a large increase in the paraxial mesoderm. Consequently the ninth placodal area comes to lie at the bottom

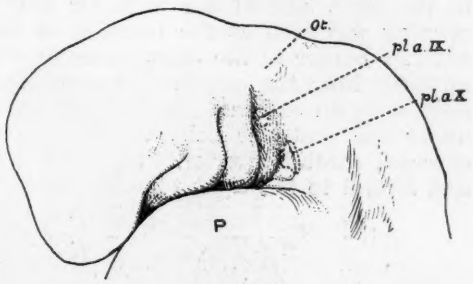

FIG. 155.—The early pharynx, showing: Ot., Otocyst; P, Pericardium; pl. a. ix., Placodal area of the glossopharyngeal nerve; pl. a. x., Placodal area of the vagus nerve. (*After J. Ernest Frazer*—'*Jour. of Anat.*', lxi.)

of a short recess which opens into the upper part of the second groove. Later the ninth placodal area is completely covered and folded in, so that it lies at the enlarged end of a deep tunnel which opens on the surface. Very soon the placodal area is completely shut off from the surface, and there is formed a buried cyst in intimate relation with the endodermal wall of the pharynx and connected with the surface by ectodermal cells (*Fig.* 156). If growth is normal, the ectodermal cyst and duct disappear at 12 mm.

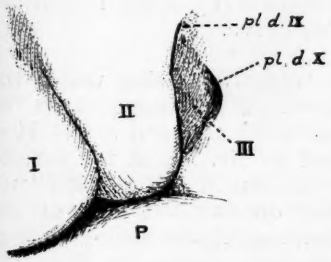

FIG. 156.—The early pharynx, showing: pl. d. ix., Placodal duct of the glossopharyngeal nerve; pl. d. x., Placodal duct of the vagus nerve; I, II, III, First, second, and third branchial arches; P, Pericardium. (*After J. Ernest Frazer*—'*Jour. of Anat.*', lxi.)

Discussing the development of this region, Frazer states, "the placodal areas certainly stand forward as the structures that are mainly affected". It is conceivable that the ninth placodal cyst lying in close relation to the second pharyngeal pouch may persist along with the duct connecting it with the surface. This is the only connection between the skin and pharynx which has been proved to exist in embryonic life. The schematic representations of internal pharyngeal ducts opening into a large covered cervical sinus from which external ducts open on the surface is incorrect and does not rest on proved facts. In the schematic representations the only structures proved to exist are the endodermal pharyngeal pouches with their endodermal ducts, and the placodal cysts with their ectodermal ducts.

The relations of the endodermal pouches in the adult pharynx are fixed. The position of the second is at the tonsil, the third in the pyriform fossa, and the fourth at the lower end and lateral aspect of the pharynx.

Lateral Pouches.—It is unjustifiable in the present state of our knowledge to dogmatize in assessing the embryological significance of vestigial structures in the neck, and in presenting three lateral pouches of the pharynx I suggest the most likely modes of origin.

Case 1.—Boy, aged 16 years, under the care of Mr. Keynes, to whom I am indebted for permission to record the case. The patient had noticed a small hole in the right side of the neck for four years. On examination a small pin-point opening was seen at the junction of the upper two-thirds and lower third of the anterior border of the right sternomastoid. A cord one inch long was felt running upwards from the orifice. At operation a cyst was found in the right side of the neck with an external opening and an internal duct passing upwards and inwards under the posterior belly of the digastric muscle and between the internal and external carotid arteries. The duct opened into the pharynx immediately below and lateral to the right tonsillar fossa. Microscopic examination of the duct close to the pharynx showed a lining of squamous stratified epithelium.

FIG. 157.—*Case* 2. Pouch on the left lateral aspect of the pharynx opening by a duct into the bottom of the left pyriform fossa. (*Specimen in the Museum of Great Ormond Street Hospital for Children.*)

In this specimen, therefore, there is a large pouch in the neck connected with the pharynx and skin by slender ducts. In embryonic life the only structures proved to connect endoderm and ectoderm in this region are the placodal cyst and duct in connection with the ninth cranial nerve. I regard this vestigial structure as a derivative of the ninth placodal cyst in relation with the second endodermal pouch.

Watson has described a similar pouch opening into the pharynx at the posterior faucial pillar whose walls were innervated by several branches from the ninth cranial nerve.

Case 2.—Infant, aged 3 weeks, under the care of Mr. Eric Lloyd. I am indebted to the Medical Committee of Great Ormond Street Hospital for Children and to Mr. Lloyd for permission to study this specimen. The child was born with a swelling on the left side of the neck which caused choking attacks during feeding. At autopsy the condition shown in *Fig.* 157 was found.

On examining the specimen I found a small round orifice at the bottom of the left pyriform fossa, and careful dissection revealed a small duct passing upwards and inwards from the cyst to the pharyngeal orifice. Microscopic examination of the cyst wall showed a lining of granulation tissue, fibrous tissue with numerous inflammatory cells, and an external muscle coat.

I regard this as a derivative of the third endodermal pharyngeal pouch and duct lying in relation with the left pyriform fossa.

Case 3.—The specimen is from the Shattock Museum of St. Thomas's Hospital, studied through the kindness of Professor Dudgeon, and is shown in *Fig.* 158. Along the left side of the pharynx and œsophagus is a thin membranous pouch 3 in. long, which communicates with the lowest part of the pharynx by a circular aperture situated in the fibres of the inferior constrictor of the pharynx, immediately behind the posterior border of the thyroid cartilage. The neck of the pouch passes behind the termination of the common carotid artery.

From the anatomical relations of this pouch, it appears to be derived from the fourth embryonic endodermal pouch. The ultimate size of the pouch is due to repeated distension with food.

Importance of the Hypoglossal Nerve in relation to Embryonic Cervical Vestigial Structures.—Frazer has drawn attention to the importance of the course of the hypoglossal nerve in relation to these structures. The hypoglossal nerve in the beginning of its course lies behind the region of the arches

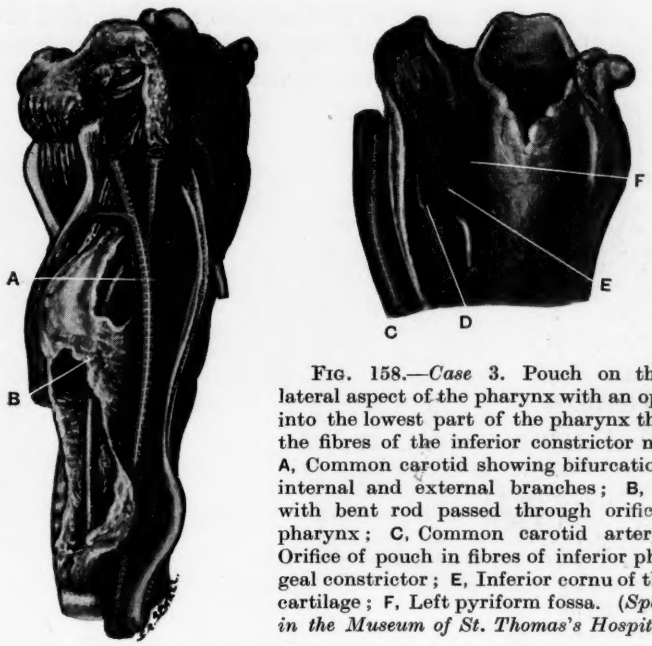

FIG. 158.—*Case* 3. Pouch on the left lateral aspect of the pharynx with an opening into the lowest part of the pharynx through the fibres of the inferior constrictor muscle. A, Common carotid showing bifurcation into internal and external branches; B, Pouch with bent rod passed through orifice into pharynx; C, Common carotid artery; D, Orifice of pouch in fibres of inferior pharyngeal constrictor; E, Inferior cornu of thyroid cartilage; F, Left pyriform fossa. (*Specimen in the Museum of St. Thomas's Hospital.*)

and grooves. In passing to its ultimate distribution the nerve is deep to structures connected with the ectoderm, and superficial to structures connected with the pharynx. Consequently any connection persisting between the surface and pharyngeal wall, or its derivatives, would be caught up over the hypoglossal nerve. On this account it is not surprising that few cervical vestiges have a deep connection, as this is usually severed by tension caused by the hypoglossal nerve.

ACQUIRED POUCHES.

Pharyngeal Diverticulum—Posterior, Acquired.—The first description of this condition is contained in a letter written in 1764 by Ludlow, of Bristol, to William Hunter. His method of examination was thorough, and included the introduction of a probang, whalebones, flexible catheters, and finally the patient was told to swallow a great quantity of quicksilver, and, as it did not enter the stomach, the surgeon was at a loss to localize it. However, the quicksilver was recovered at post-mortem from a large pharyngeal pouch.

Sir Charles Bell in 1816 first shed light on the etiology, by calling attention to spasmodic contraction at the œsophageal entrance followed by repeated

ineffectual attempts to swallow. Zenker and Von Ziemssen in their classical study of 1877 contributed to the morbid anatomy, symptomatology, and diagnosis of the condition.

ETIOLOGY.—Many theories have been advanced in regard to the pathogenesis of the condition. There is no evidence that the structure is atavistic in origin. Some authorities have likened it to the pouch which occurs in the domestic pig, but the morphology of this structure is entirely different. Various erroneous congenital theories have been advanced. It may be stated at once that the pouch is a prolapse of the mucous membrane of the pharynx in an area bounded by two different sets of musculature, innervated by different nerves. A consideration of the structure of this area surprises one that the condition is not more frequent.

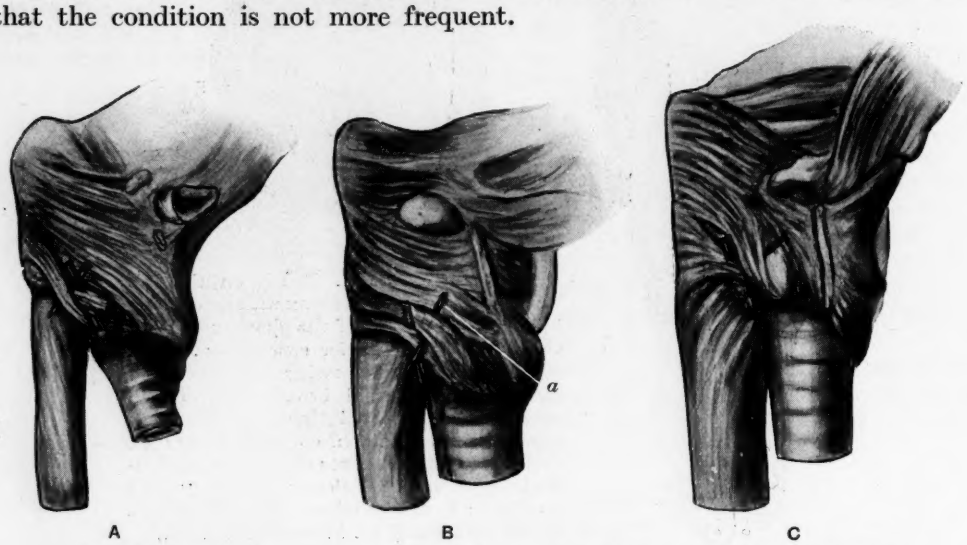

FIG. 159.—The arrangement of the fibres of the cricopharyngeus muscle in monkeys. A, The orang: the muscle is composed of superficial constrictor fibres; there is a special sphincteric muscle arising from the thyroid cartilage (red rod). B, The chimpanzee: the white rod (*a*) is under the superficial constrictor fibres; the red rod is under the deep sphincteric fibres. C, The silver gibbon: arranged as under B.

The Œsophageal Orifice in Different Species.—In the dogfish the œsophagus is not more than 3 in. in length and entirely composed of a muscular sphincter of strong circular fibres. In the crocodile the cornua of the hyoid bone guard the œsophageal entrance and form a well-marked sphincter. This mechanism is complicated still further in the tortoise. In mammalia the hyoid bone no longer controls the œsophageal entrance; the cricoid cartilage now forms the basis of the sphincteric mechanism. Negus has shown that such a mechanism is necessary in all lung-breathing forms to prevent air suction into the œsophagus during respiration. The ornithorhyncus has a well-marked band of muscle encircling the œsophageal entrance, forming a sphincter with a weak attachment to the fused cricothyroid ring. The arrangement of the muscles in monkeys is shown in *Fig.* 159.

In the human fœtus the cricopharyngeus muscle is divided into an upper superficial constrictor part, incorporated in the pharyngeal constrictor musculature, and a lower deeper sphincteric part which blends posteriorly

with the muscle of the œsophagus. In a four-months' fœtus (*Fig.* 160A) this division is evident, and the constrictor portion passes obliquely upwards whilst the sphincteric part passes transversely round the œsophagus. In a five-months' fœtus (*Fig.* 160B) the direction of the sphincteric portion is obliquely downwards. Consequently a weak area is produced in the posterior wall of the pharynx between the superficial and deep portions of the cricopharyngeus muscle. It is through this weak area that the pharyngeal mucosa prolapses in the adult (*Fig.* 161). A fully formed pouch is shown in *Fig.* 162.

I have satisfied myself in examining a large number of these pouches that the mucous membrane of the pharynx only herniates through this area.

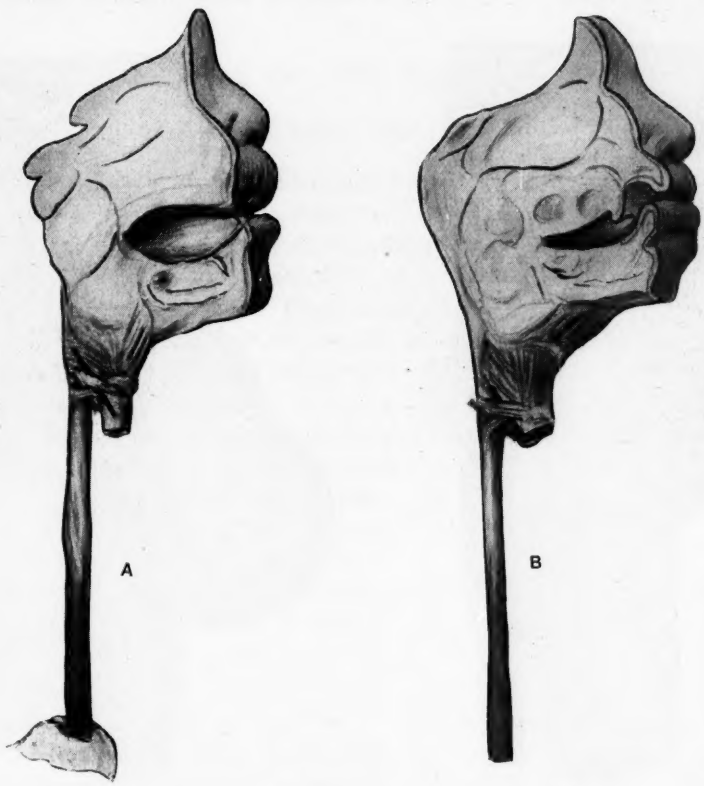

FIG. 160.—The arrangement of the cricopharyngeus muscle in the fœtus. A, Four months; B, Five months. The black rod is under the superficial constrictor fibres; the red rod is under the superficial constrictor and deep sphincteric fibres.

The cricopharyngeus muscle therefore consists morphologically and functionally of two distinct sets of musculature with a congenital weakness between them. If either of the two following conditions is present, prolapse of the mucous membrane will occur.

1. Loss of elasticity or degeneration of the muscles composing the lower pharyngeal constrictors. At best, this musculature is poor material, and even under normal conditions this region is one of stress and strain.

2. Persistence of contraction of the sphincteric portion of the cricopharyngeus muscle during deglutition is probably the more important factor.

We know this occurs from the histories of patients with pouches. Consequently the intrapharyngeal pressure increases and the weakest part of the wall gives way.

During the second stage of normal deglutition the pharyngeal constrictors contract, and simultaneously the sphincteric portion of the cricopharyngeus muscle relaxes. This is a complex and involuntary reflex action. Why the sphincter fails to relax in certain individuals we do not know. It is feasible to suggest that it is due to a breakdown in the neuromuscular mechanism. We are familiar with such breakdowns in certain other regions of the alimentary canal, but as yet we do not know the site of the primary lesion, whether it is central, ganglionic, peripheral, or due to some internal secretion.

FIG. 161.—Early prolapse of pharyngeal mucosa between the two parts of the cricopharyngeus muscle. (*Specimen in the Museum of the Royal College of Surgeons.*)

FIG. 162.—Pharyngeal pouch. The two parts of the cricopharyngeus muscle are seen. (*Specimen kindly lent by Mr. Cawthorne.*)

The nerve-supply around the œsophageal entrance is complex. The pharyngeal constrictors, including the constrictor portion of the cricopharyngeus, are supplied by the intricate pharyngeal plexus. The upper end of the œsophagus, including the sphincteric part of the cricopharyngeus, is supplied by numerous motor nerves from the recurrent laryngeal nerves. Contributions to the innervation of this region are also made by the sympathetic system, but the rôle of these nerves is obscure. The association of goitre with this type of pouch has been noted by German writers and is of interest in connection with derangement of the neuromuscular mechanism. It has

been suggested that the enlarged thyroid gland raises the intrapharyngeal pressure by pressing on the œsophagus and thus herniation of the pharyngeal mucosa occurs. It is more likely, however, that the motor nerves to the œsophageal orifice are mechanically stimulated by the enlarged thyroid gland, with consequent spasm of the sphincter. I suggest the possibility of œsophageal spasm in these cases being due to increased excitability of the vago-sympathetic nerves to the sphincter, caused by deranged thyroid metabolism.

Pharyngeal Pouch—Anterior, Acquired.—This type of pouch is very rare. Hurst and Briggs have recorded an example in a female aged 57 years which was discovered by radiological methods immediately in front of the entrance to the œsophagus and behind the larynx.

POUCHES OF THE ŒSOPHAGUS.

CONGENITAL POUCHES.

Pouch Associated with an Œsophago-tracheal Fistula.—The marked constancy of the morphology of this condition has been recognized by numerous observers and is a sign of an early fundamental change in the embryo, as changes occurring late in embryonic life give rise to anomalies which do not conform to a definite pattern. There may be a genetic basis for this malformation, as Mackenzie found it present in all the children of one father by three wives.

The pathological picture is very constant (*Fig.* 163). The œsophagus commences in the usual way and ends blindly, forming a uniformly dilated pouch with thin walls. The large size of the pouch may be due to distension with amniotic fluid or to an abnormal growth stimulus. In most cases the lower portion of the œsophagus opens into the trachea at the bifurcation or a short distance above.

Numerous theories have been put forward to account for this anomaly. Malformation of the œsophago-tracheal septum has been widely accepted, but as we do not know precisely how the œsophagus and trachea separate, the theory does not satisfy. Others have advanced theories of intra-uterine inflammation and intra-uterine trauma.

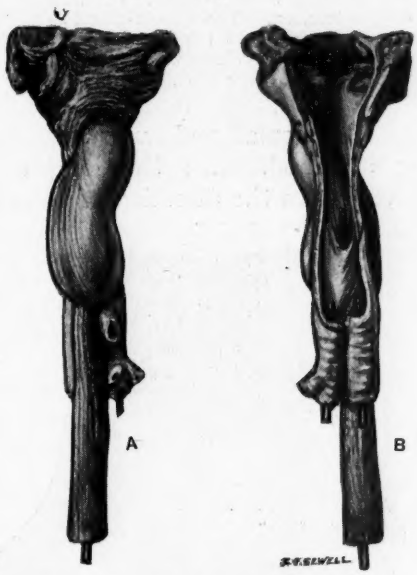

FIG. 163.—Œsophago-tracheal fistula associated with a pouch of the upper end of the œsophagus. A, Posterior aspect; B, Anterior aspect.

It is not surprising that congenital anomalies should occur sometimes, when we consider the intricacies of the developmental process, each species negotiating the various stages at specific rates, which vary within certain normal limits. If these normal limits are transgressed, the product of development is distorted. Stockard has performed experiments in which the rate

of development was modified at will and various congenital malformations were produced. He showed that temporary arrest of growth at certain critical moments in development is followed by disastrous results. Stockard states that practically any deformity recorded in the literature, other than those resulting from germinal variations or mutations, may be induced by lowering the temperature, which modifies the rate of growth. The malformation under review, therefore, may be due to changes in the rate of growth in the cells which normally separate the trachea from the œsophagus. The exact nature of the forces causing these changes is little understood at present. Changes in temperature and environment may play a prominent part.

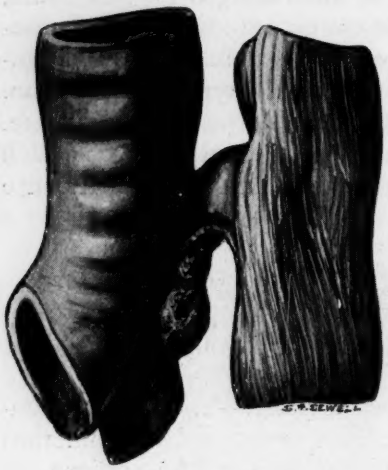

FIG. 164.—Pouch in anterior wall of œsophagus above the bifurcation of the trachea. The wall of the pouch is intimately incorporated in the wall of the trachea. The lymph-gland is merely attached to the lower border of the pouch. (*Specimen in the Museum of the Royal College of Surgeons.*)

Œsophageal Pouch due to Imperfect Separation of the Trachea and Œsophagus.—Pouches of this nature occur in the anterior wall of the œsophagus a short distance above the bifurcation of the trachea or in the angle of bifurcation. Careful comparison with pouches due to adherent tuberculous lymph-glands show marked differences. Whereas pouches due to inflamed lymph-glands are small and taper towards the apex, to which a mass of lymph-glands are attached, with the long axis placed obliquely upward or downward according to the direction of the glandular pull, the pouch under consideration

FIG. 165.—Pouch in anterior wall of œsophagus with the upper wall incorporated in the wall of the trachea. There is a depression in the right bronchus corresponding with the fundus of the pouch. (*Specimen in the Museum of St. Bartholomew's Hospital.*)

is round, with a broad fundus and circular orifice, with the long axis at right angles to the œsophagus, or obliquely upward towards the trachea or bronchus (*Figs.* 164, 165). Moreover, a part of the wall of the pouch adjoining the trachea is firmly incorporated in the tracheal wall. The muscle bundles are clearly demarcated on each side of the pouch.

If development is pursuing a normal course, the œsophagus and trachea separate at 5 mm. and the musculature of the tubes is formed at 10 mm. Hence the primary defect in the wall must be epithelial. In this connection Ribbert has demonstrated primitive ciliated cylindrical epithelium in the walls of these pouches.

In a series of transverse sections of a 17-mm. human embryo kindly lent to me by Professor J. E. Frazer, there is imperfect separation of the trachea and œsophagus with epithelial connection between the two tubes in three sections (*Fig.* 166). Such a defect may manifest itself in adult life as an œsophago-tracheal fistula, or partial closure may occur and the epithelium of the œsophagus become incorporated in the wall of the trachea at the site of the original fistula. Further development of the pouch is due to tension at the site of union due to elongation of the œsophagus, a certain degree of rotation which the œsophagus undergoes when the stomach rotates, and longitudinal contraction and relaxation of the whole œsophagus.

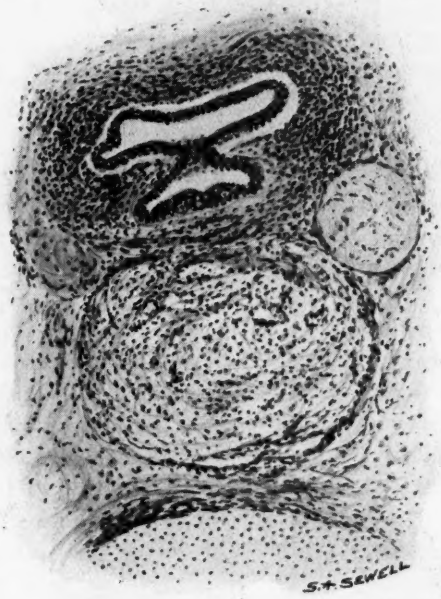

FIG. 166.—Transverse section of 17-mm. human embryo showing epithelial connection between the trachea and œsophagus. The position of the œsophagus is shown by the solid cord of cells. (*Serial sections of an embryo kindly lent by Professor J. E. Frazer.*)

Œsophageal Pouch Associated with Defect in the Muscular Coats.—According to Keith the œsophagus is of double origin: the upper or paratracheal part is derived with the trachea from the retropharyngeal segment of the foregut, and the lower or retropharyngeal part arises from the pregastric segment of the foregut.

I have noticed the marked difference in the upper and lower halves of the œsophagus in animals, the former being largely a membranous tube and the latter a strong muscular tube. In a four months' fœtus the muscle coat of the lower half is well developed, whilst the upper half remains membranous. In the main, the muscle is unstriated in the lower half and striated in the upper half. Oppel regards the striated muscle as derived from the branchial muscle which arises from the lower head myotomes.

Fig. 167 shows a pouch of this nature occurring immediately below the bifurcation of the trachea, composed entirely of mucous membrane which does not bulge. The muscular defect is round, with clean-cut edges encircling

FIG. 167.—Pouch in anterior wall of the œsophagus below the bifurcation of the trachea.

FIG. 168.—Two small pouches below the bifurcation of the trachea. (*Specimen in the Museum of St. Bartholomew's Hospital.*)

FIG. 169.—Œsophageal pouch associated with multiple diverticula of the colon. (*Specimen in the Museum of Charing Cross Hospital.*)

the pouch. The two lymph-glands have no connection with the pouch. In *Fig.* 168 there are two small pouches immediately below the bifurcation of the trachea; the other coats of the œsophagus are absent.

It is obvious that this type of pouch is not due to traction or pulsion, and I consider it to be due to a primary muscular defect in the region of union between muscle derived from the lower head myotomes and unstriated muscle developing *in situ.* Such muscular defects have been described in embryos of various ages. Happich found that the circular muscle in embryos of three and four months was completely interrupted in small areas. Shridde found a larger muscular defect in the œsophageal muscle in contact with the trachea.

Œsophageal Pouch Associated with Multiple Diverticula of the Colon (*Fig.* 169).—The association of œsophageal diverticula with those in other regions of the alimentary canal has been noted by Morrison and Smelt. The view is gaining ground that acquired diverticula of the whole intestinal tract from the œsophagus to the anus owe their origin to similar tendencies and are local variations of the same morbid process. Barsony and Polgar have described multiple œsophageal diverticula which they consider to be due to disturbed innervation.

Keith regards colonic diverticula as the manifestation of irregular contractions of the circular muscle of the colon due to disturbed innervation. This may be true for the colonic and certain multiple œsophageal diverticula, but it is difficult to apply this theory to the example under review. Lewis and Thyng have demonstrated knob-like intestinal diverticula occurring regularly in embryos of man, rabbit, and pig, which usually degenerate but sometimes persist, in various regions. In connection with this specimen it is reasonable to suppose the presence of such congenital defects with the manifestation of well-marked pouches in later life.

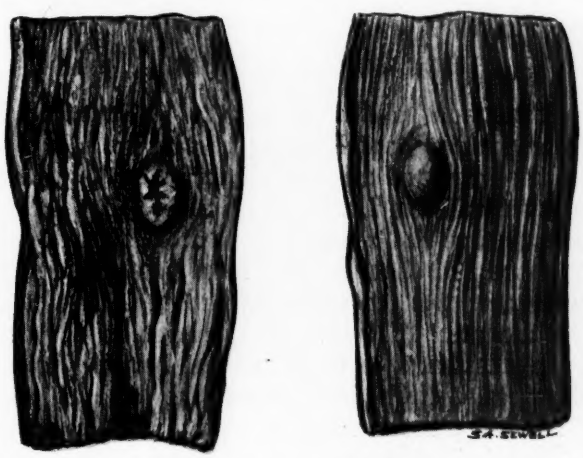

FIG. 170.—Pouch in the posterior wall of the œsophagus. (*Specimen in the Museum of St. Bartholomew's Hospital.*)

Pouch in the Posterior Wall of the Œsophagus.—Pouches in the posterior wall of the œsophagus are rare, and the appearances differ from all we have already discussed (*Fig.* 170).

The character of the pouch suggests that it may have originated in a cyst which has acquired a lumen into the œsophagus. Cysts of the œsophagus probably arise early in embryonic life from two sources. In human embryos about 20 mm. vacuoles of various sizes occur in the œsophageal epithelium. In some places they open directly into the lumen, in others they are separated from it by a partition of epithelium. The vacuoles are more numerous in the lower half of the tube and in the anterior and posterior walls. Vacuolation increases the size of the lumen of the œsophagus. These epithelial cavities may persist, forming either cysts or diverticula.

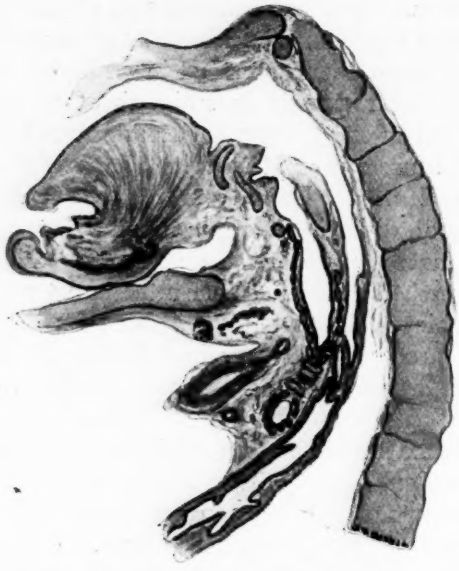

FIG. 171.—Diagram reconstructed from serial sagittal sections of a 10-weeks embryo, showing the developing œsophagus.

In later embryonic life the epithelium of the œsophagus is markedly irregular (*Fig.* 171). In circumscribed areas epithelial proliferation is taking place and epithelial bridges across the lumen occur. In other areas there is an active moving apart of the cells, resulting in well-marked depressions. These depressed areas may be isolated from the main lumen and covered in by proliferating epithelium. Such cavities may remain closed, forming cysts, or may secondarily acquire an opening into the œsophagus, forming a diverticulum.

ACQUIRED POUCHES.

Œsophageal Pouch due to Adhesion with Tuberculous Lymph-glands.—Kragh has made important contributions to the pathology of this type of pouch and prefers the term 'tuberculous diverticulum'. The pouch is conical in shape with an oval orifice with the long axis obliquely upward or downward. The muscle coat of the œsophagus seldom stops at the margin of the diverticulum. The apex of the pouch is firmly attached to diseased lymph-glands. The commonest site for their occurrence is in the anterior wall of the œsophagus in the area following the bifurcation of the trachea, where the œsophagus is in relation with the bronchial lymph-glands (*Fig.* 172).

Adhesions between lymph-glands and the œsophageal wall are not frequent; Kragh found adhesions in 14 out of 556 cases examined.

This type of pouch is uncommon in children. Dr. Allan Brown kindly searched the records of the large Children's Hospital in Toronto for me and could find no example. Mediastinal tuberculous lymphadenitis is more common in Toronto than in this country.

Pouches Associated with Obstruction at the Lower End of the Œsophagus.—Pouches associated with cardiospasm are found in two regions—namely,

FIG. 172.—Pouches in the œsophagus due to lymphadenitis. (*Specimen in the Museum of the Royal College of Surgeons.*)

FIG. 173.—Fusiform pouch associated with stricture of the terminal œsophagus in a child. (*Specimen in the Museum of Great Ormond Street Hospital for Children.*)

FIG. 174.—Large pouch in the lateral wall of the œsophagus. The lumen of the lower end of the œsophagus is much diminished. (*Specimen in the Museum of the Royal College of Surgeons.*)

between the fibres of the cricopharyngeus muscle and in the lower third of the œsophagus. Twelve cases of pharyngeal pouch secondary to cardiospasm have been reported in the literature. Dessicker believes that a history of cardiospasm may be elicited in the majority of cases of pouches in the lower third of the œsophagus.

Pouches due to an organic stricture at the lower end of the œsophagus are fusiform or saccular. In the former the œsophagus is dilated throughout a localized area proximal to the stricture (*Fig.* 173). Abel has recorded an example of a saccular pouch situated above a stricture.

FIG. 175.—Pouch in the right lateral segment of the œsophagus. (*Specimen in the Museum of St. Bartholomew's Hospital.*)

The effects of a stricture at the lower end of the œsophagus may be either uniform dilatation of the whole œsophagus, or partial dilatation with the formation of a fusiform pouch, or the muscle bundles may diverge in a localized area with consequent herniation of the mucous membrane (*Fig.* 174).

Œsophageal Pouch of Inflammatory Origin.—The relation of pouches and ulcers has excited interest. Percy and Shaw have drawn attention to the connection in the duodenum. *Fig.* 175 shows a diverticulum in the right postero-lateral segment of the œsophagus 4 in. above the cardiac orifice, with a wide oval orifice whose long axis is in the line of the œsophagus. The overlying muscle is absent and there is evidence of œsophagitis. It appears that this pouch is of inflammatory origin and very probably originated in an ulcer.

NATURAL POUCHES IN ANIMALS.

The Sloth Bear.—There are two nasopharyngeal pouches, which Mayer described in 1830 (*Fig.* 176). According to Killian, they are not homologous with any structure in the pharynx of man.

Domestic Pig.—The nasopharyngeal pouch of this animal was described by Mayer in 1840. It is a large sac passing downward and backward from the lower end of the nasopharynx (*Fig.* 177). The orifice is guarded by a prominence formed by the lower part of the plica pharyngopalatini. The apex commonly projects backwards between the two divisions of the

FIG. 176.

FIG. 176.—Nasopharyngeal pouch of the sloth bear. (*Specimen in the Museum of the Royal College of Surgeons.*)

FIG. 177.—Nasopharyngeal pouch of the domestic pig.

FIG. 178.—Anterior pharyngeal pouch of the great anteater.

FIG. 179.—Upper and lower œsophageal pouches of the fruit bat.

FIG. 177.

FIG. 178.

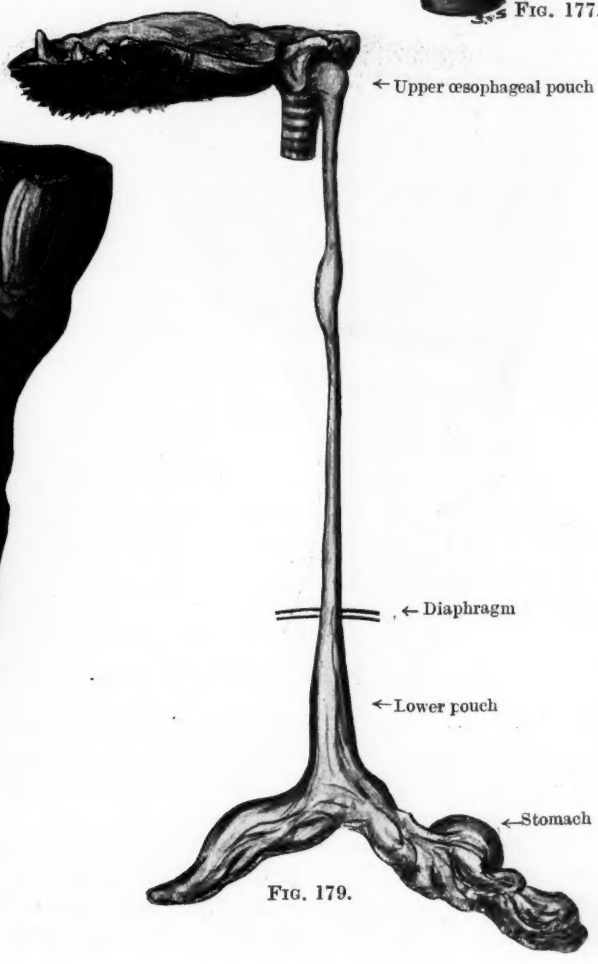

FIG. 179.

thyropharyngeus muscle. The upper part of this muscle acts as a loose sling, and the lower part is firmly blended with the inferior border of the pouch. An oblique muscle on each side passes deep to the thyropharyngeus to an attachment at the apex of the pouch. There is thus concerted muscular action at the apex to prevent excessive increase in size. The lower part of the thyropharyngeus muscle is divided into superficial and deep portions continuous above, and there is no space through which a pharyngeal pouch could occur as in man. It is clear that the morphology of this pouch is entirely different from the posterior pulsion pouch in man.

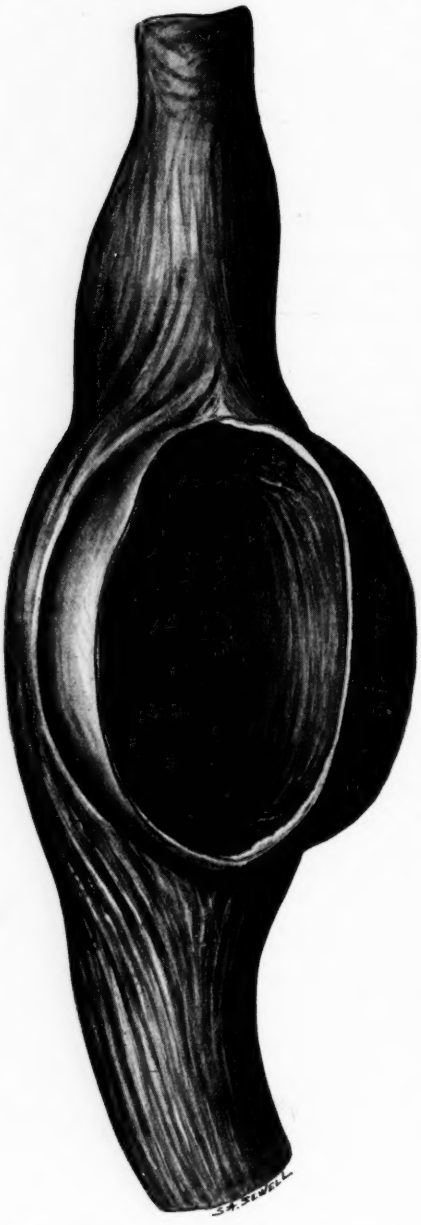

FIG. 180.—Large œsophageal pouch in a horse. (*Specimen in the Museum of the Royal College of Surgeons.*)

FIG. 181.—Pouch at lower end of œsophagus in a dog, containing a foreign body. (*Specimen in the Museum of the Royal Veterinary College.*)

Pharyngeal Pouches.—These are uncommon in animals. *Fig.* 178 shows an anterior pharyngeal pouch which I removed from a great anteater. The membranous sac projects upwards and forwards above the hyoid bone. The orifice lies in the pharynx above the base of the epiglottis. It is difficult to

assign a function to this pouch. It is certainly not for the storage of food, and I suggest it secretes mucus for the lubrication of the food passages.

Œsophageal Pouches.—In the fruit bat I found a pouch at the upper end of the œsophagus which has escaped the notice of earlier observers (*Fig.* 179). The pouch was present in three animals I examined, and consisted of a rounded sac situated between a tight circular sphincter mechanism above and weaker constrictor muscle fibres below. This animal spends many hours hanging head downwards from trees, and since there is no sphincter at the lower end of the œsophagus, fluid runs up the tube, but is prevented from entering the pharynx by the strong sphincter at the junction of pharynx with œsophagus. Hence the pouch is a pressure pouch, and the manner of production, lying as it does between a tight sphincter and a weak constrictor, is remarkedly similar to the mechanism of production of the pressure pouch of man. Immediately below the diaphragm there is a fusiform pouch which is morphologically part of the stomach.

Pathological Pouches in Animals.—Pathological pouches in the pharynx of animals are rare, as they are not systematically looked for. In monkeys there is no reason why they should not occur, except in the orang, where the lower end of the pharynx is strengthened by a specialized muscular band. Negus has recorded a pharyngeal pouch occurring in a mangabey ape.

Pathological pouches commonly occur in the œsophagus of animals. The horse is specially prone to pouch formation owing to rupture of the muscle coats (*Fig.* 180). Pouches occur frequently in the dog (*Fig.* 181).

I wish to thank the Council for the privilege of delivering this Arris and Gale Lecture. My best thanks are due to Mr. Burne for much kindness and help and to Mr. Wilson. I gratefully acknowledge the help of Professor G. E. Gask, Colonel Argyle, Sir Thomas Dunhill, Professor J. Ernest Frazer, Mr. Carwardine, Mr. Capps, Dr. Finzi, Dr. Sparks, and Dr. Simon, and the Curators of many hospital museums.

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THE SURGICAL TREATMENT OF BRONCHIECTASIS.*

By ROBERT M. JANES, Toronto.

Attempts to relieve cases of bronchiectasis by surgical intervention have been, on the whole, discouraging. A fresh stimulus to the study of the disease was given by the introduction of lipiodol by Forrestier in 1922: before the employment of this diagnostic agent it was impossible, in the majority of cases, to be certain of the existence of bronchiectasis, and the accurate localization of the disease was out of the question. In a series of 72 cases Hartrung (quoted by Hedblom[1]) was able by ordinary methods and röntgenograms to make a correct diagnosis as to the presence and site of the lesion in only 20; in 39 it was impossible to recognize either the lesion or its location. Under such circumstances great surgical progress was not to be expected.

Surgical methods of treatment may be divided into three main groups: (1) Collapse therapy as obtained by pneumothorax, section or exeresis of the phrenic nerve, or thoracoplasty; (2) Pneumotomy with drainage of the larger collections of pus; and (3) Excision or destruction of the involved lung tissue.

Collapse Therapy.—The pathological picture presented by the disease would seem to make it obvious that no form of collapse therapy could accomplish more than an alleviation of symptoms. The extent of the area in which secretions can collect may be decreased, but the underlying infection must still persist in the large majority of cases, and in very few is there a marked permanent reduction in the calibre of the dilated bronchi.

Oakley[2] recorded the results of *phrenic paralysis* in 17 cases of bronchiectasis. Complete relief of symptoms was reported in 4 cases (23·5 per cent), lasting improvement in 7 (41 per cent), and temporary improvement with relapse in 6 (35·5 per cent). Hedblom[1] reported 38 cases in his own experience, in which following phrenicotomy 9 were free of symptoms for from three months to three years, 20 had a decrease of 50 to 80 per cent in the amount of sputum, 7 showed some improvement, and 2 were unimproved. Davies[3] produced a unilateral phrenic paralysis in 25 cases of basal bronchiectasis. Ten (right side) were not improved, and thoracoplasty was proposed; 5 (left side) obtained symptomatic relief and coughed less, but their condition was reported as worse eighteen months later; 1 (right side) was completely free of all symptoms for two years and then had recurrence with hæmoptysis and death. In a recent report[15] he states that of 5 cases in which

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* From the Department of Surgery of the University of Toronto and the Toronto General Hospital. Based on the work of the author and his senior colleague, Dr. N. S. Shenstone. Read at the combined meeting of the Canadian and Ontario Medical Associations at Toronto, June 24, 1932, as the surgical contribution to a symposium on bronchiectasis.

Table I.—Results of Seven Cases of

Case	Sex	Age	Location	Duration	Admission	Operation
1. H. H.	M.	32	R.L., R.M.	1 year ..	June 21/28	Phrenic neurectomy Oct. 9/28
					Feb. 27/29	Multiple-stage extrapleural thoracoplasty between March 6 and April 19/29
2. A. M.	M.	27	L.U. and L.	1 year, 3 months	June 18/30	Multiple-stage extrapleural thoracoplasty between July 30 and Oct. 21/30
					Sept. 19/31	
3. A. W.	F.	24	L.U. and L.	Well until 10 days before admission	Feb. 15/30	Thoracotomy Feb. 25/30
				13 months ..	March 12/31	Multiple-stage extrapleural thoracoplasty between April 1 and May 20/31
4. J. C.	M.	43	L.U.* ..	1 year, 8 months	Jan. 26/32	Drainage of abscess Feb. 11/32
						Thoracoplasty 1–4 posterior April 2/32
						5–9 posterior April 23/32
5. H. H.	M.	50	R.U. ..	12 years ..	July 2/32	Upper right posterior thoracoplasty July 22/30
6. H. W.	M.	43	L.U. and L.	16 years ..	June 6/32	Multiple-stage thoracoplasty June 8/32 to Aug. 30/32
7. R. D.	M.	24	L.U. and L.	8 years ..	Aug. 8/32	Multiple-stage thoracoplasty Aug. 11/32 to Oct. 13/32

* Bronchiectasis occurred as complication of lung abscess.

BRONCHIECTASIS TREATED BY THORACOPLASTY.

ANÆSTHESIA	DISCHARGE OR DEATH	COMPLICATIONS	RESULTS
	Jan. 28/29		
Intercostal nerve-block	May 21/29	Septic pneumonia left lower lobe with large abscess	Became worse during period of operative treatment. Had extension to left lower lobe with formation of abscess. At autopsy right lung fibrous throughout and contained almost no pus
Spinal or intercostal nerve-block for posterior. Gas and oxygen for anterior	Nov. 9/30 Oct. 7/31	Carcinoma of bronchus ..	In course of thoracoplasties sputum gradually decreased from 12 to 14 oz. daily to a maximum of 1 oz. daily. Following discharge Nov. 9/30 health improved for 7 or 8 months and then began to suffer from attacks of dyspnœa. Upon re-admission Sept. 19/31 found to be still in fair general health with little sputum. Died. Autopsy showed carcinoma the size of a walnut at entrance to left primary bronchus
	Aug. 1/30	Empyema	Patient admitted with widespread bronchopneumonia left side. Empyema occurred which was drained. Went on to a widespread bronchiectasis. Transferred to sanatorium, where a gain of 20 lb. in weight occurred but sputum remained unchanged
Spinal for lower. Gas and oxygen for upper and anterior	Aug. 8/31	Nil	Patient developed a small amount of bronchitis of opposite side. Stood operations well, but there was little decrease in sputum. Not much improved When heard from several months later had gained somewhat in weight and improved in general health. Sputum only slightly decreased
Gas and oxygen ..	June 13/32		Upon discharge from hospital general health good. Sputum consisted of a little clear mucus only. Drainage wound almost healed. No discharge
Gas and oxygen			March 1/33. No cough, no sputum
Gas and oxygen ..	July 29/30	Bronchopneumonia left side	Post-operative course at first fairly satisfactory, temperature to 101°. Died July 29. Autopsy showed an extensive saccular bronchiectasis of the right upper lobe, adherent pleura right side. Bronchopneumonia on left side apparent cause of death
Spinal, gas and oxygen, or local	Sept. 12/32	Mild phlebitis left femoral	March 20/34. Looks well. Cough and sputum still troublesome, but sputum 2 to 4 oz. daily as compared with maximum of 20 oz. daily before operation, and less foul
Spinal, gas and oxygen, or local	Nov. 21/32	On Oct. 25/32 developed a widespread bronchopneumonia in the right lower and middle lobes, gradually recovering from this. On discharge there were only a few râles at the right base. Sputum 4 to 8 oz. daily and foul	March 16/33. Has gained 30 lb. since leaving hospital. Sputum, about 20 oz. before operation, now about 4 oz. daily, but still very foul. Has had no further pneumonia

R.L. = Right lower. R.U. = Right upper. R.M. = Right middle. L.U. and L. = Left upper and lower.

the operation was done in the hope that it might be self-sufficient, 4 gained no benefit though the fifth was relieved of symptoms for two years. In two other cases in which the evulsion was done as a preliminary to thoracoplasty, the major operation had been postponed indefinitely on account of the improvement resulting from the hemidiaphragmatic paralysis. In our experience the relief obtained by paralysis of the diaphragm has not been worth while.

Pneumothorax has not been used by us in the treatment of the disease. Several of our cases had been treated in this manner, however. In some the sputum had been decreased temporarily, in others no noticeable improvement had occurred. Where adhesions do not prevent its use we believe it should be employed pre-operatively as an aid in emptying the dilated bronchi of their infective contents and perhaps lessening the danger of pneumonia. We consider, however, that, except perhaps in early cases in children, artificial pneumothorax should be used only as a pre-operative measure, since, if the air is allowed to absorb, the subsequent development of adhesions renders the operative procedure more difficult: such adhesions also inhibit the proper expansion of the upper lobe, preventing the obliteration of the cavity left after operation and increasing the likelihood of a bronchial fistula.

The number of cases which we have treated by *thoracoplasty* is too few to form the basis of an opinion. The results obtained may be seen in *Table I*. *Case* 1 was one of the rapidly progressive type. Although he died shortly after the completion of the collapse, the condition of the compressed lung at autopsy suggested that, had the infection of the opposite lung not occurred, marked improvement might have been expected. *Case* 5 also had an extension to the good side. *Case* 2 should, of course, never have been operated upon, and illustrates the necessity for a bronchoscopic examination. The decrease in sputum and improvement in general health were, however, sufficient to have made the operation well worth while. *Case* 3 was improved, but not to the extent that might have been expected. *Case* 4 is well. *Cases* 6 and 7 are moderately improved at present, but too a short time has elapsed to permit a final judgement.

In 1931 Hedblom[1] reported that of 14 cases that had had a thoracoplasty before 1926, 3 had remained symptom-free, 7 raised from 10 to 20 per cent less sputum: 6 of these were working regularly, 4 who were improved died from ten months to two years after operation—one of actinomycosis. Of 18 cases operated upon since 1926, 4 died (one of brain abscess, one of pulmonary gangrene, and two of pneumonia). The remaining 14 were all improved, with sputum reduced from 60 to 90 per cent; 2 died following a secondary lobectomy. In view of the results that may be obtained by lobectomy it would seem that extrapleural thoracoplasty should be reserved for those cases of unilateral bronchiectasis in which the area of involved lung is too great to be removed completely. The degree of collapse required is much greater than in pulmonary tuberculosis. Because of the large amount of sputum, local or spinal anæsthesia is preferable, and the operation should be done in multiple stages, usually at least four.

Drainage.—The drainage of one or more of the large abscess pockets would appear to have been used extensively by Sauerbruch. He recommends

its employment[4] and states that it often results in considerable alleviation of symptoms but never in cure. The pathology of the condition makes it clear that little could be expected from simple drainage. We have not attempted to treat bronchiectasis *per se* by drainage, but have consistently failed to obtain a cure by drainage alone in those cases of chronic lung abscess in which a secondary bronchiectasis had developed.

LOBECTOMY.

That lobectomy would be the ideal method of treatment of localized bronchiectasis has been recognized by most writers on the subject. Surgeons have been loath to advocate it because of the tremendous mortality associated. Lilienthal reported a personal mortality of 47 per cent. Coryllos[5] in 1930 collected 87 cases operated upon by eleven different surgeons, with 48 deaths—a mortality of 53·1 per cent. Of the surviving cases, 38 were cured by operation and 1 was improved. Coryllos reported 2 of his own cases, 1 cured without a fistula, 1 cured apart from the presence of a fistula. He advocated a preliminary graded thoracoplasty followed by lobectomy in one or two stages; one lobe was removed with a cautery. Whittemore[6] described a method in 1927 in which under gas and oxygen anæsthesia a sufficient number of ribs were removed to permit of the diseased lobe being freed and brought to the surface, where it was firmly fixed to the chest wall by deep sutures of silk and chromic catgut. In about ten days the lung tissue became necrotic, and in four to five weeks it sloughed away, leaving a clean granulating wound, which was lightly packed until it closed. The cases were in hospital for an average of six to eight weeks, and the wound required from three to four months to close. He did not do a preliminary thoracoplasty, but suggested that it might be wise to do so. He reported 6 cases, 3 of which were healed without fistula: one was well but had a persistent fistula at the end of one year and three months; one was still in hospital; and one died on the tenth day from pneumonia on the good side.

Robinson[7, 8] described a method of two-stage lobectomy. At the first sitting the overlying ribs, periosteum, and intercostal bundles were removed. At the second stage the lobe was freed, clamped at the pedicle, and excised. Individual vessels were ligated, and finally a mass ligature of silk or kangaroo tendon was placed on the pedicle. The cavity was packed and allowed to granulate. He reported 5 cases with 1 death[8], and stated that the resulting bronchial fistulæ were easily closed by skin- and fat-grafts. In his other paper he reported 7 cases with 3 deaths. Archibald[10] successfully performed a two-stage lobectomy in 1924.

Graham[9] advocated the cautery excision of the diseased lung. Part of the overlying chest wall was first removed and the wound packed widely open. The diseased tissue was destroyed later in multiple stages with a cautery iron. He reported 20 cases, of which 4 (20 per cent) were completely healed, 6 (30 per cent) were free of symptoms but had bronchial fistulæ, 3 (15 per cent) showed marked improvement but still had some cough, 2 were still in process of treatment, 1 had not been heard from, 4 (20 per cent) were dead.

Hitzrot[11] reported a successful one-stage lobectomy in 1920. The chest was opened through an intercostal incision in the seventh interspace; the lobe was freed from dense adhesions and removed. The intercostal wound was closed completely and drainage established by the Kenyon method through an intercostal stab-wound. A bronchial fistula developed, but closed spontaneously. A complete cure was accomplished. McDowell[12] reported a single successful case operated upon by Tudor Edwards in 1930. The chest was closed tightly and the pleural effusion, which remained sterile, aspirated upon ten occasions. The patient, a boy of 16, became completely well. That the effusion would remain sterile in many cases could not be expected.

TECHNIQUE.

The technique which we have developed is a modification of that employed by Brunn[13]. It was described by Shenstone in a paper given before the conjoined meeting of the British and Canadian Medical Associations in Winnipeg, on Aug. 27, 1930.

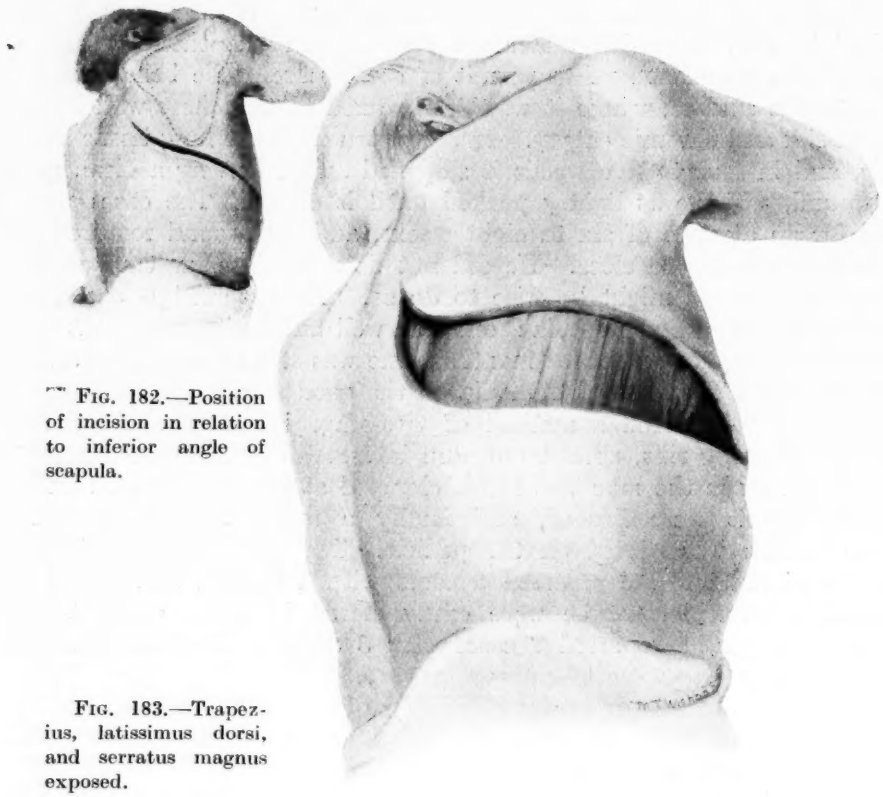


FIG. 182.—Position of incision in relation to inferior angle of scapula.

FIG. 183.—Trapezius, latissimus dorsi, and serratus magnus exposed.

A long incision is made in the general direction of the ribs, passing just below the scapula and extending from a point a few inches above and behind the angle of that bone to the costal cartilages in front (*Figs.* 182, 183).

The underlying muscles are severed in the same direction (*Fig.* 184), exposing the ribs and intercostal spaces. We have usually chosen the sixth interspace as our portal of entry into the chest, though the fifth or seventh appears to be equally satisfactory. The subsequent exposure is much enlarged by

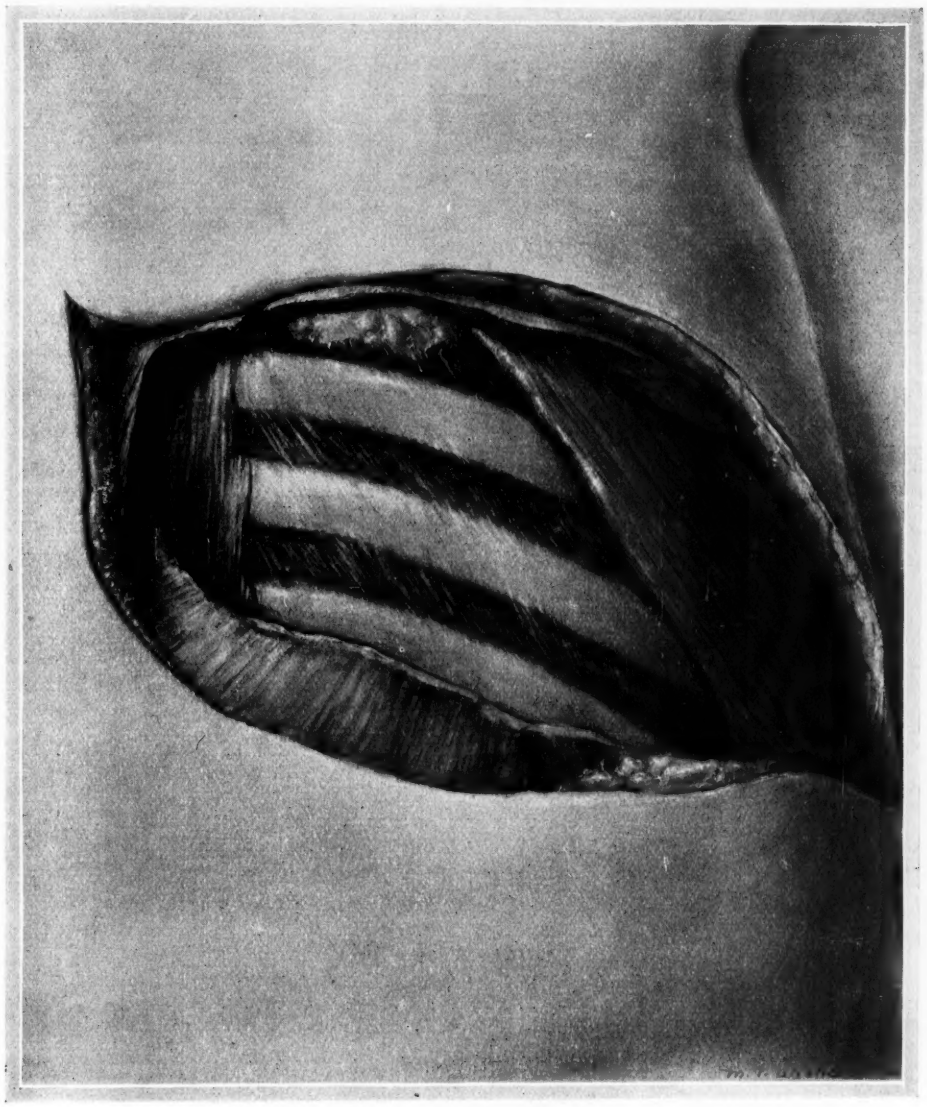


FIG. 184.—Division of trapezius and latissimus dorsi muscles, exposing erector spinæ and serratus anterior muscles, 6th, 7th, and 8th ribs.

dividing the ribs adjacent to the selected interspace as near their vertebral ends as possible, and this we have done in each case before entering the thorax (*Fig.* 185). For this purpose the lateral margin of the erector spinæ is dissected medially, and the tip of a straight bone-cutting forceps inserted close to the rib, which is severed without prior elevation of the periosteum.

In the earlier cases the intercostal muscles were divided carefully and air allowed to enter slowly by plugging the opening intermittently with the tip of the finger (*Fig.* 186). We now feel that cases with a free pleura should have a preliminary pneumothorax. If this has been done, no special precautions are necessary. The intercostals with the underlying pleura may be divided safely from the costal cartilage to the angle of the ribs and the

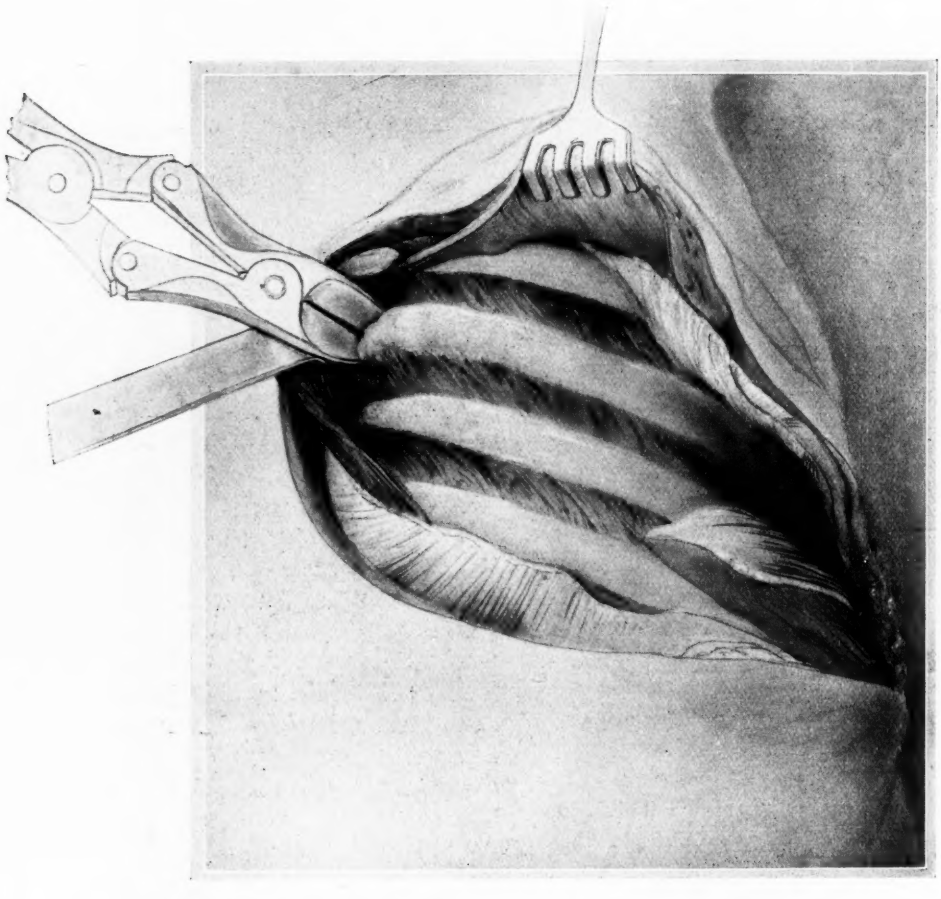


FIG. 185.—Elevation and retraction of the lateral border of the erector spinæ muscle, permitting the section of the 6th and 7th ribs near their vertebral extremity.

wound spread widely with self-retaining retractors (*Fig.* 187). An enormous exposure is obtained in this way.

Adhesions are variable: in our experience they are always present in the interlobar fissure, and usually so between the lower lobe and the diaphragm. In several of our cases the pleural space was obliterated. Adhesions are separated by a combination of sharp and blunt dissection, and great care must be taken to avoid wounding the lung.

When the diseased lobe has been freed completely a snare of heavy cord is passed around it as near as possible to the mediastinum, and the loop drawn tight in the instrument designed for the purpose (*Fig.* 188). We believe

it to be a better method of controlling the pedicle than any other we have seen described, and nothing has given us such a feeling of comfort and safety in this operation as this simple device. Without the devitalization of the tissue, it controls the blood-supply, obstructs the bronchi, provides a solid support during the suture of the pedicle, and, in addition, can be used in limiting mediastinal movement during the operation.

FIG. 186.—Intercostal muscles divided: 6th and 7th ribs retracted. Tip of finger placed over opening in pleura to ensure slow entry of air into pleura. (*After Brunn.*)

For the purpose of preventing the escape of infected material during the section of the lung, a second snare is placed about $1\frac{1}{2}$ in. distal to the first in those cases in which the diseased lobe is sufficiently voluminous (***Fig.*** 189). When the lobe to be resected has become very small as a result of extensive fibrosis or collapse, this second snare has been found to interfere

with the making of a properly excavated pedicle and its use has been discontinued. The general pleural cavity is then protected by moist packs and the lobe cut away, leaving a pedicle ¾ in. in length. The section is best accomplished with curved scissors, and the cut surface of the pedicle should be somewhat excavated to assist in the subsequent closure (*Figs.* 190–193).

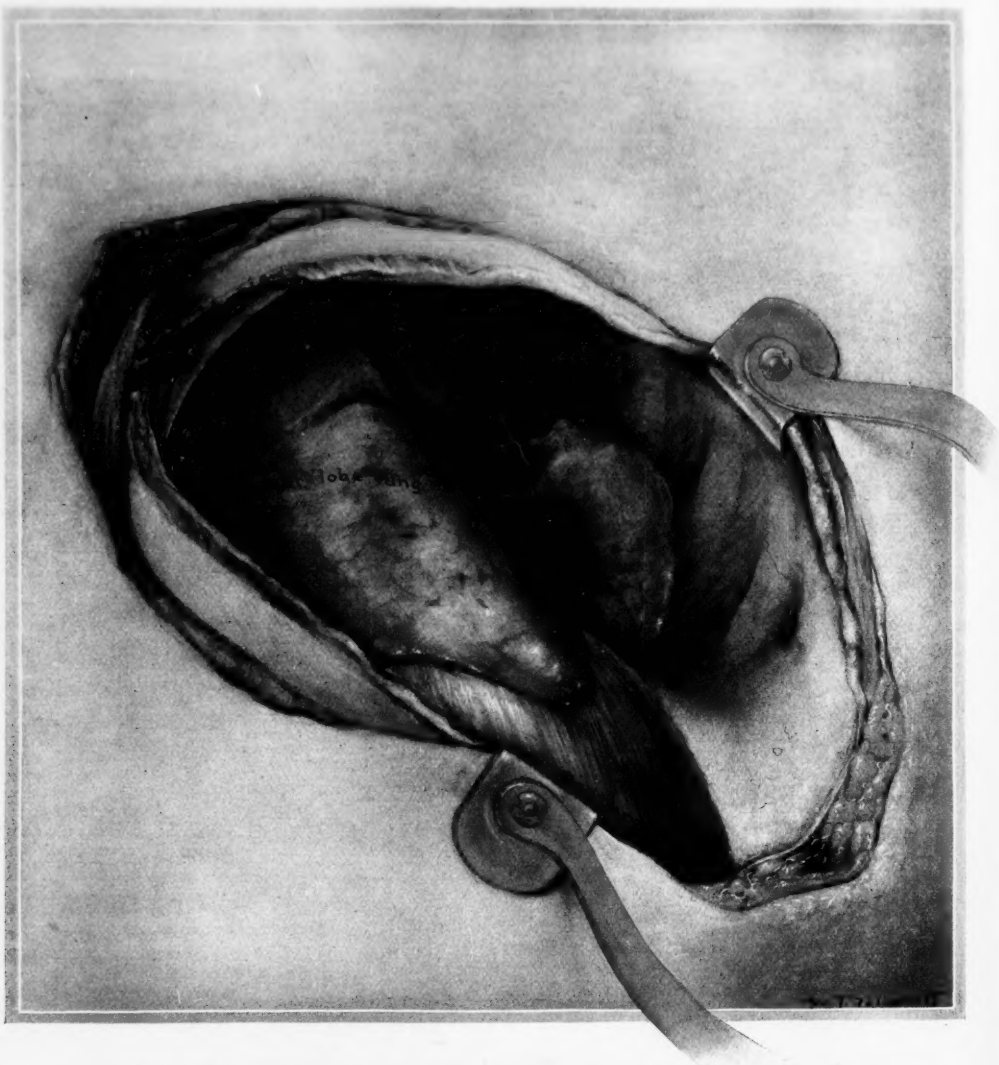


FIG. 187.—Exposure obtained in a case without pleural adhesions.

Frequently one or more bronchi project above the surface: these should be trimmed away, but care should be taken that a bronchus with a bronchial artery does not slip back out of the snare, as this may result in the vessel being missed by the running suture and subsequent hæmorrhage into the bronchus. The resultant bare area is painted with acriflavine (1–1000).

Obvious vessels are caught with forceps and ligated, and a running suture of catgut is introduced across the pedicle but not including its pleural margin. The snare is then slightly released and any oozing areas can be included in the suture before it is tied. A further running catgut stitch closes over the pleural margins, an endeavour being made to invert the edges in the process. The snare is then removed and the remaining lobe carefully examined for any small tears overlooked earlier.

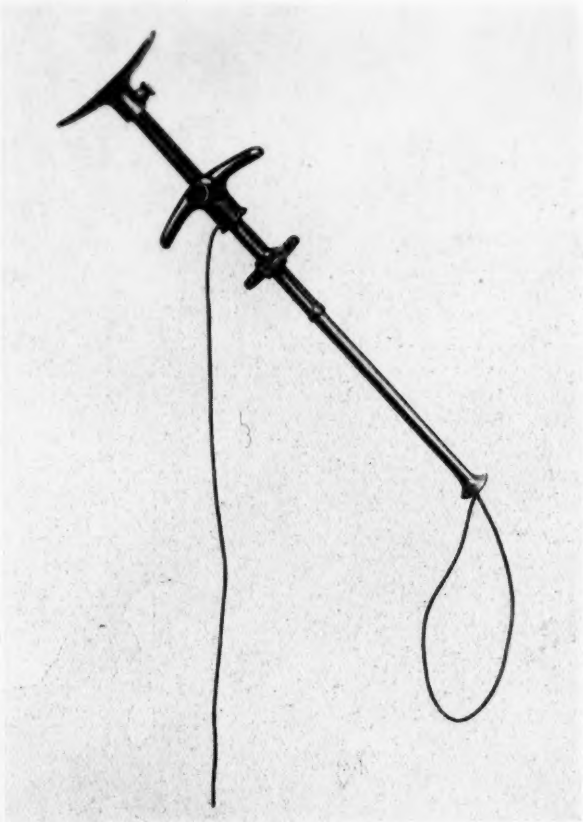


FIG. 188.—Special tourniquet designed to control the pedicle.

It is important to have decided from lipiodol injection before operation whether or not there is an involvement of the middle lobe on the right or the lingual portion of the upper lobe on the left side. We have found that inspection and palpation of the lung through the open chest cannot be relied upon. The middle lobe is occasionally sufficiently pedunculated to be freed and removed along with the lower. More often it must be dealt with separately, and is then removed with a snare in the same way as the lower lobe, or, where it cannot be freed to a definite pedicle, it must be clamped and removed in the same manner as a portion of the upper lobe.

When it is desired to remove part of a lobe, two crushing clamps are applied proximal to the diseased area and the lung sectioned between them.

The cut surface is painted with acriflavine. A running suture of chromic catgut is then placed over the forceps, the forceps removed, and the suture pulled taut. The bare area is covered in with a continuous Lembert suture. A small incision is then made in the ninth interspace in front of the mid-axillary line through which the end of a long tube of about 32F calibre is drawn. The fenestrated end of this tube is placed about 1 in. away from

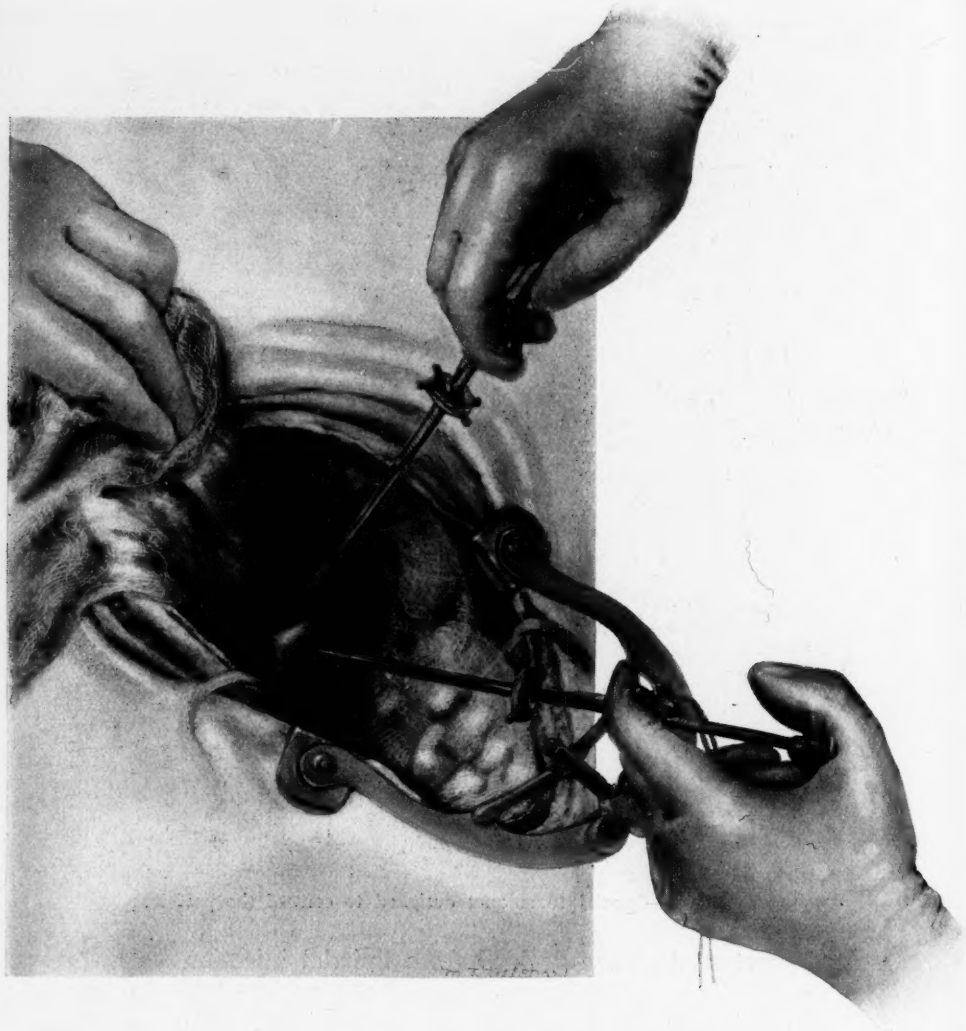


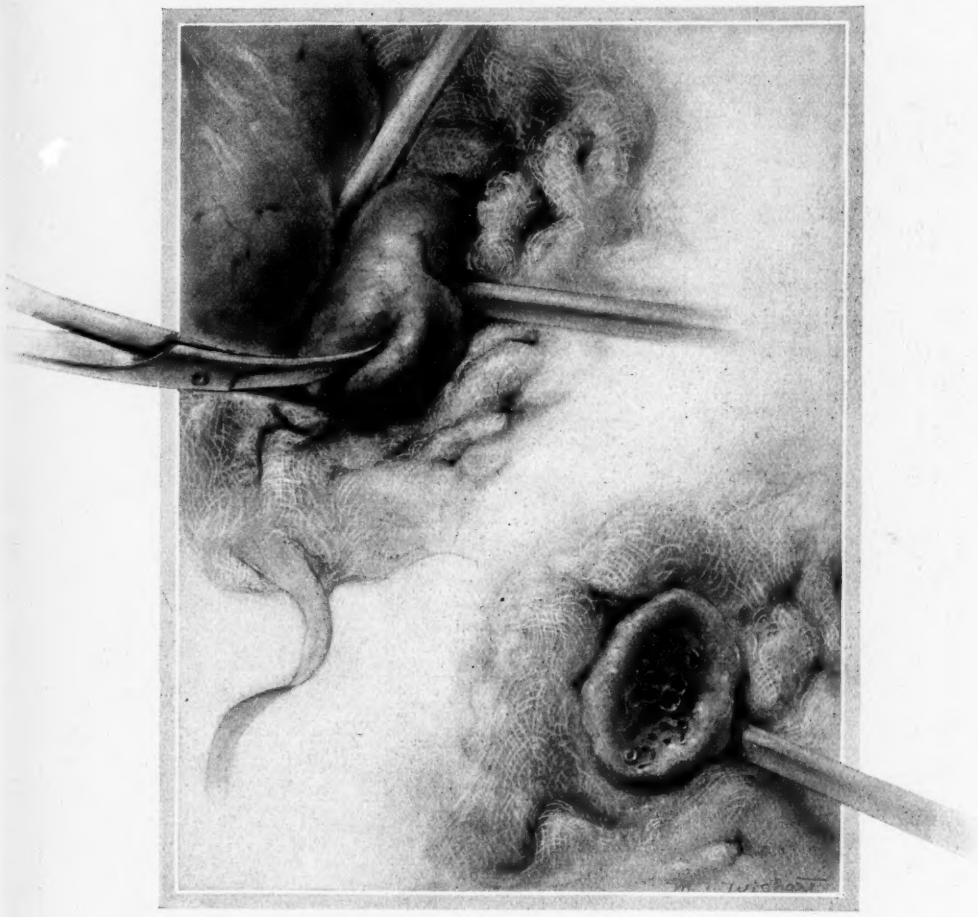
FIG. 189.—Tourniquets in place. Exposed surfaces protected with gauze pads.

the pedicle of the resected lung and maintained in place by a plain catgut suture inserted into the summit of the diaphragm (*Fig.* 194).

The operation wound is closed in layers. Interrupted chromicized catgut sutures enclosing the adjacent ribs bring the edges of the long rent in the intercostals easily and accurately together (*Fig.* 195). In our experience the inclusion of the nerves has not caused any lasting pressure neuritis. The

muscles are approximated by a continuous suture of chromicized catgut and the skin by an ordinary dermal suture.

As soon as the wound is closed, the distal end of the drainage tube is placed under the surface of a normal saline solution contained in a bottle, maintained at least 2 ft. below the level of the chest. The patient is then invited to cough. This act expresses the air from the pleural cavity, permits of the rapid expansion of the lung, and prevents mediastinal flapping. At the end of twenty-four hours it has been our custom to attach a continuous

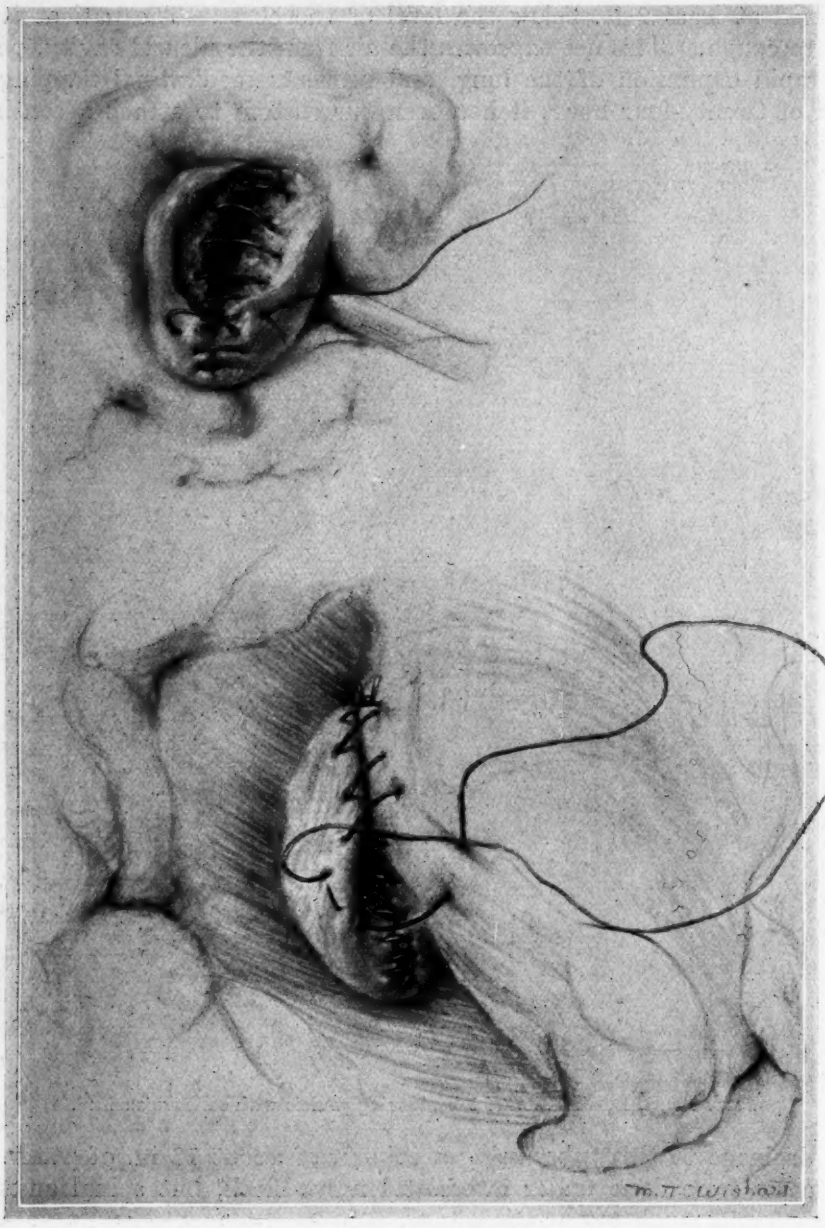


FIGS. 190, 191.—Ablation of lobe, leaving pedicle with concave surface.

siphon drainage to this tube, using a column of about 18 in. of water. The earlier cases were done under intercostal nerve-block, but spinal anæsthesia has been found much more satisfactory.

The use of the snare has made it easy to deal with the pedicle. There is no loss of blood, no escape of air, and much less spread of infected material. The flapping of the mediastinum may be entirely controlled. Apart from the fact that the placing of sutures is made so much easier, the patient experiences great comfort from this support and coughs much less frequently.

Under spinal anæsthesia there is a minimum of shock and it has been possible to proceed without hurry. The patient is able to cough and clear a considerable amount of the material expressed into the trachea. Flooding of the



FIGS. 192, 193.—Closure of pedicle and its subsequent suture to upper lobe.

opposite lung is not avoided entirely, however, and the occurrence of pneumonia in the good lung remains the greatest hazard. One case developed a massive collapse of the opposite lower lobe and subsequently

a bronchiectasis: this was probably due to the aspiration of tenacious sputum into that bronchus.

FIG. 194.—Drainage tube in place.

The patient is placed in bed in the Fowler position. Post-operative respiratory embarrassment and cyanosis is variable; in most cases it is slight, but occasionally is very considerable. It is often necessary to administer

oxygen until the pressures have been readjusted, and no patient should be operated upon unless an oxygen tent or oxygen chamber is available. Repeated hypodermic injections of atropine $\frac{1}{100}$ gr. help to control the œdema, but this does not seem to be nearly so effective as the administration of oxygen. Fairly frequent changes of position should be insisted upon. The

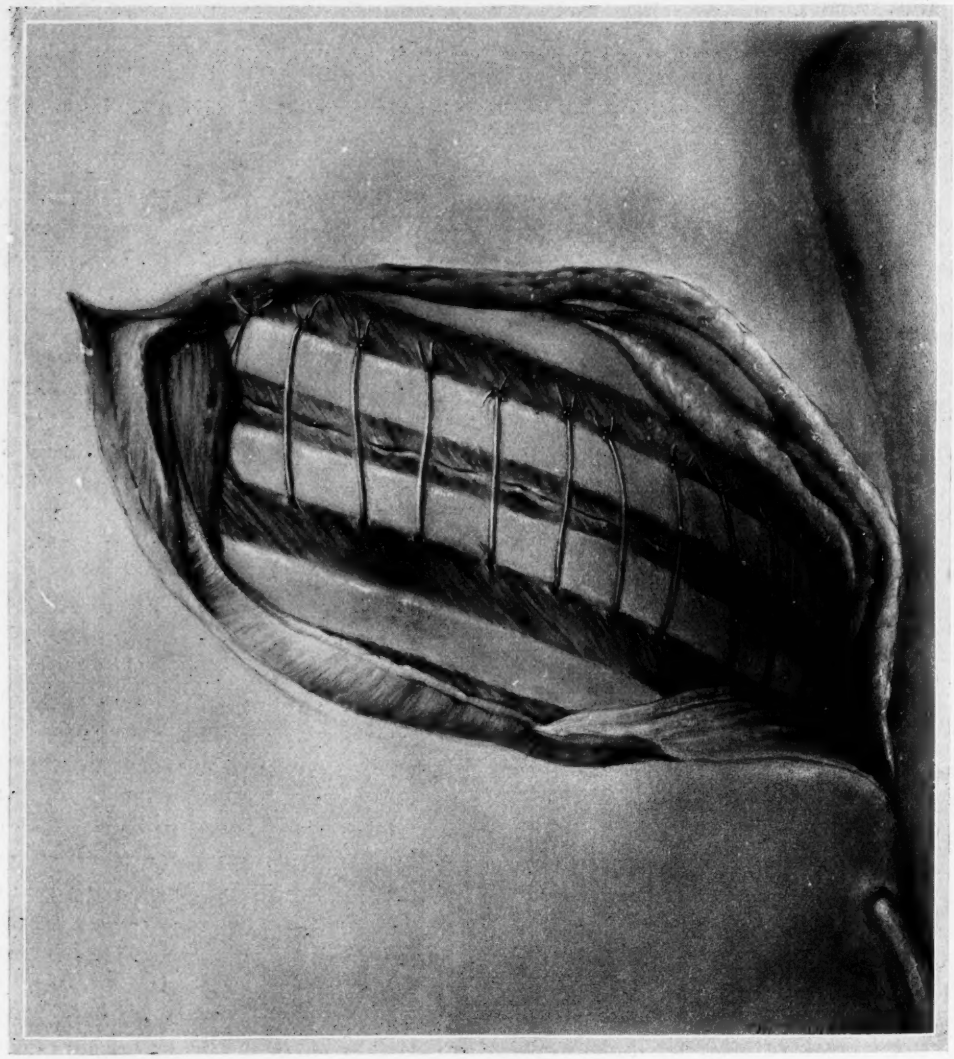


FIG. 195.—Method of closure: medial suture includes erector spinæ muscle.

patient should be encouraged to cough and thus clear bronchial secretions. Morphine should be administered in $\frac{1}{6}$-gr. doses sufficiently frequently to ensure reasonable comfort and to permit of coughing without too much pain.

For the first twenty-four hours there is a considerable escape of blood and serum. If the upper lobe is free from adhesions, it is displaced downward and quickly becomes adherent to the elevated dome of the diaphragm.

It is this application of the upper lobe to the diaphragm as well as the method of closure of the stump that prevents the formation of a bronchial fistula, and we have noted that if the descent of the upper lobe is prevented by pleural adhesions, a fistula is more likely to occur. In many of our cases there has occurred a temporary pneumothorax in the upper pleura in which a variable amount of fluid has been present. Both air and fluid may be absorbed rapidly, the lung expanding to fill the space. In two of the cases this condition was accompanied by infection requiring drainage through intercostal stab-wounds. In one of these rapid healing took place; in the other a partial thoracoplasty was required for closure of a persistent cavity. More recent experience has convinced us that collections of fluid above the upper lobe should be drained as soon as recognized. An X-ray should be made with the patient in the sitting position within the first two or three days following the operation, as it is not always possible to interpret correctly the clinical findings. If drainage is established promptly, infection may be prevented, any embarrassment to respiration is removed, the lung is allowed to expand more rapidly, and the cavity is much more likely to close spontaneously.

Two cases developed increasing fever for which no cause could be discovered up to the tenth day post-operatively, at which time they suddenly coughed up several ounces of pus. The X-ray of one such case showed a definite cavity with a fluid level about the stump of the resected lung. In both cases cough and sputum ceased at the end of four to six weeks. They are both well and apparently cured. This complication prolongs convalescence, but since the abscess is efficiently drained into a main bronchus it would appear that spontaneous recovery could be anticipated.

SUMMARY OF RESULTS.

An analysis of *Table II* reveals the following points of interest.

Of 17 cases operated upon, 16 were done by the one-stage method. Of these 16 cases, 6 were cured, 3 were improved, 2 unimproved, and 5 died. In one case (*Case* 5) improvement only (and not cure) occurred from failure to recognize an involvement of the lower portion of the upper lobe on the same side; in another (*Case* 9) owing to the presence before operation or possibly the development after operation of some disease in the opposite lobe. One (*Case* 14) developed a post-operative collapse of the previously healthy opposite lower lobe, in which there is now a well-developed bronchiectasis. The two unimproved cases were more extensive than was realized. Complete post-mortem examinations were obtained upon the 5 cases that died. Two deaths (*Cases* 4 and 8) were found to have been due to bronchopneumonia in the previously normal lung of the opposite side. *Case* 16 had a large amount of sputum, but lipiodol injection seemed to demonstrate that the bronchiectasis was confined to the left lower and lower part of the left upper lobe. Autopsy showed that death had been due to bronchopneumonia, empyema, and suppurative pericarditis; the remaining portion of the left upper lobe and right lower lobe were bronchiectatic. A cavity the size of a hen's egg in the upper lobe had failed to fill with lipiodol. This was very disturbing, and demonstrated that even the lipiodol injections cannot be depended upon

entirely for localizing the disease. Had the extent of the lesion been appreciated, one would not have thought of attempting even a thoracoplasty. There was no evidence that the pericardium had been injured at operation and no apparent reason for the occurrence of the pericarditis. The fourth case (*Case* 11) died of secondary hæmorrhage from a perforating branch of the internal mammary artery eight months after the primary operation and three weeks after a persistent bronchial fistula had been closed, apparently successfully, by a plastic operation. This death should be looked upon, probably, as an unfortunate accident, since the diseased lung had been removed entirely, but is a reminder of the fact that no case in which a fistula is still present can be regarded as out of danger. The fifth case died of a brain abscess fifty-two days after operation.

Of the 16 cases, 6 developed bronchial fistulæ. Three of these were temporary only and closed spontaneously, one required an operation for closure (*Case* 11), and in two (*Cases* 16, 17) the patient failed to survive. When the upper lobe is freely movable and becomes adherent to the dome of the diaphragm over the drainage tube, the fistula, should one form, consists of a long, narrow tract and closes readily. When, however, an empyema forms between the upper lobe and the diaphragm and the large bronchus opens directly into this, spontaneous closure is not usual and a plastic operation may be necessary later. It is interesting that of 12 left-sided cases operated upon, only 3 developed fistulæ; of these, the one in the patient that survived closed readily. On the other hand, of 4 right-sided cases operated upon, 3 developed fistulæ, one of which was closed by operation.

Of the 16 cases, 4 developed more or less extensive empyemata. Two of these were above the upper lobe; one closed readily following intercostal drainage; one was closed by thoracoplasty. Two formed between the upper lobe and diaphragm; the one in the patient that survived was closed by thoracoplasty. (*Table II.*)

Massive collapse of the opposite lobe occurred once only, but proved a serious complication, since a bronchiectasis subsequently developed.

It is essential, before considering possible therapeutic measures, that the location and the extent of the disease be determined as accurately as possible. The usual physical examination of the chest and ordinary radiographs do not give the definite evidence that is necessary, and, in all cases, most careful injections of the bronchial tree with lipiodol must be made, especial attention being given to the condition of both lower lobes, the inferior part (lingual lobule) of the left upper, and the right middle lobes. Even with the utmost care in the injections an occasional diseased bronchus is missed.

These examinations having been carried out, we have accepted for surgical consideration cases limited to one lower lobe with or without involvement of the lingual lobule on the left side or the corresponding middle lobe on the right.

We have considered the operation justifiable in those patients whose sputum was large in amount or foul, in those who had suffered numerous pulmonary hæmorrhages whether or not they had sufficient sputum to be

Table II.—Results of Lobectomy in Bronchiectasis.

Case	Sex	Age	Date of Admission	Date of Operation	Character of Operation	Date of Discharge or Death	Post-operative Days in Hospital	Temporary Fistula	Complications	Result
Two-stage Lobectomy—										
1. M. S.	F.	34	May 22/28	Jan. 22/29 Mar. 6/29	R.L.	Mar. 11/29	5 died	No	Bronchopneumonia	Died
One-stage Lobectomies—										
2. M. R.	F.	19	May 3/29	May 4/29	R.L. and R.M.	June 9/29	36	Yes	Osteomyelitis of rib	Cured
3. A. D.	M.	47	Nov. 24/29	Nov. 30/29	L.L.	Dec. 23/29	23	No	—	Cured
4. C. C.	F.	20	Aug. 20/29	Dec. 18/29	R.L. and R.M.	Dec. 23/29	5 died	Yes	Bronchopneumonia	Died
5. R. L.	F.	26	Nov. 5/29	Feb. 21/30	L.L.	May 29/30	99	No	Bronchopneumonia	Improved
6. V. W.	F.	23	Feb. 17/30	Mar. 26/30	L.L. and part of L.U.	June 30/30	96	No	Empyema (U) drainage	
7. J. S.	M.	30	Feb. 12/30 June 30/30	July 2/30	L.L.	Apr. 17/30 Oct. 29/30	89	No	Empyema (U) drainage and thoracoplasty	Unimproved
8. L. S.	M.	52	Aug. 31/30	Oct. 8/30	R.L.	Oct. 14/30	6 died	No	Bronchopneumonia	Died
9. D. McG.	F.	14	Oct. 24/30	Nov 1/30	L.L.	Dec. 17/30	46	No	Pleurisy and acute bronchitis ..	Improved
10. K. F.	F.	21	Jan. 21/31	Feb. 11/31	L.L.	May 5/31	83	No	Secondary infection of wound	
11. F. L.	M.	22	Jan. 28/31	Feb. 27/31	R.M. and R.L.	Oct. 30/31	245 died	Yes	Empyema and secondary hæmorrhage	Died
12. B. O'B.	F.	24	Mar. 25/31	Apr. 1/31	L.L.	June 18/31	78	No	—	Unimproved
13. M. K.	F.	20	July 28/31	Aug. 19/31	L.L. and part of L.U.	Oct. 1/31	43	No	—	Cured
14. M. C. S.	F.	30	Aug. 14/31	Aug. 19/31	L.L.	Oct. 31/31	63	No	Massive collapse of right lower lobe ..	Improved
15. G. H.	F.	32	Nov. 23/31	Dec. 9/31	L.L.	Jan. 21/32	43	Yes	—	Cured
16. J. T.	M.	24	Apr. 19/32	Apr. 27/32	L.L. and part of L.U.	May 27/32	38 died	Yes	Bronchopneumonia, pericarditis, and empyema	Died
17. M. L.	F.	17	Apr. 26/32	Apr. 30/32	L.L.	June 21/32	52 died	Yes	Brain abscess ..	Died

R.L. = Right lower. R.M. = Right middle. L.L. = Left lower. L.U. = Left upper.

in itself significant, and in those in whom the disease appeared to be definitely progressive.

A review of our series has demonstrated that cases with persistent fever and those recently recovered from exacerbations of the disease are unsuitable for operation. We feel that a considerable afebrile interval should elapse before lobectomy is advised in these cases, and that the procedure is probably safer if carried out during the warmer weather of the year. The post-operative septic pneumonias following operations where these obvious precautions are not taken are of a peculiarly serious type and have been the immediate cause of death in 40 per cent of our fatal cases. Patients having a large amount of sputum should carry out postural drainage for some time before operation, and we prefer to operate upon them in the later, rather than in the early, part of the morning. If the sputum is foul, and particularly if numbers of spirochætes are demonstrable, it is possible that one or two administrations of one of the intravenous preparations of arsenic should be given before operation.

It is a pleasure to acknowledge my indebtedness to Dr. Shenstone for the privilege of reporting this work and for much helpful criticism in the preparation of the paper, and to Miss M. T. Wishart for the patience and care with which she has prepared the illustrations.

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PULMONARY LOBECTOMY.*

TECHNIQUE AND REPORT OF TEN CASES.

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THE technique of the operation we are about to describe is in all essential details that of Shenstone and Janes,[1] of Toronto. In 1931 one of us was in Toronto and had the advantage of seeing these surgeons perform their operation and of following the post-operative course; and this account is based on the experience of our first ten consecutive cases operated on at the Brompton and St. Bartholomew's Hospitals. We differ from them in some minor details of technique and have introduced several new instruments which we consider add to the facility of the operation.

Until quite recently lobectomy has had a mortality of about 50 per cent. The chief difficulties were a generalized infection of the pleural cavity, the closure of the pedicle after removal of the lobe, mediastinal infection, and mediastinal emphysema. On account of the danger of a total empyema many surgeons perform the operation in two or more stages. In the absence of adhesions Lilienthal[2] takes steps at the first stage to promote adhesions over the upper lobe either by rubbing the pleura with dry gauze or painting it with iodine. John Alexander[3] does this as a routine at the first stage of his lobectomy operation. Some surgeons emphasize the danger of a large open pneumothorax in the absence of adhesions or rigidity of the mediastinum, but from our operative experience we are convinced that this is a false doctrine. During the one-stage operation the mediastinum certainly does shift towards the opposite side, but without any appreciable embarrassment to the patient's pulse or respiration. To overcome the difficulties of closure of the bronchial stump and pleural infection, Sauerbruch,[4] Whittemore,[5] John Alexander,[3] Zaaijer,[6] and others advocate strangulation of the diseased lobe either by ligatures or a rubber band, following which the diseased lobe is drawn outwards to occlude the opening in the chest wall (exteriorization), and in the course of the next two weeks sloughs off. In order to obliterate the space left after the lobectomy, and also to promote adhesions, Harrington,[7] Hedblom,[8] and Coryllos[9] advocate a thoracoplasty beforehand, but this is entirely unnecessary, because if the remaining lobe is free, it has no difficulty in expanding to fill the entire hemithorax.

*Submitted for publication, January, 1933.

In 1929 Harold Brunn[10] pointed out that no serious infection of the pleura occurred in a one-stage operation if early expansion of the remaining lobe was encouraged. By closing the chest tightly and employing a negative pressure drainage he showed that the lobe became adherent before the discharge became purulent. Brunn's idea stimulated Shenstone and Janes to further work on the one-stage technique. Their operation differs from Brunn's only in the method of closing the pedicle. The one-stage operation in a free pleura is now shown to be at least as safe a procedure as the 'strangulation' operation, and has the advantage that there is no permanent diaphragmatic paralysis and that the remaining lobe is not crippled by dense adhesions and deformity of the chest wall.

ESSENTIAL PRINCIPLES OF THE OPERATION.

1. The entrance to the thorax through a long intercostal incision, with the result that there is no permanent deformity of the chest wall.
2. The freeing of adhesions not only round the diseased lobe but also over the costal surface of the remaining lobe so that it will be free to expand and fill the hemithorax after the operation.
3. The employment of two tourniquets on the pedicle of the lobe, the proximal one to control bleeding and the distal one to prevent escape of infected material while the lung is amputated between these two.
4. The closure of the stump by a continuous suture rather than by dissection and separate ligation of its component structures. This we feel is an essential point in prevention of mediastinal infection or emphysema.
5. Negative pressure drainage to remove any fluid and encourage the early expansion of the upper lobe, in order to obliterate all the dead space and so reduce the infected area to a small track.

PRE-OPERATIVE CONSIDERATIONS.

Indications.—As the object of this paper is to describe a technique, we do not wish to dwell at length on the indications or contra-indications for lobectomy. It is obviously an ideal treatment for a chronic disease which is confined to one only of the lobes of the lung. Bronchiectasis, either cystic, atelectatic, or acquired, is the condition for which the majority of operations are performed. The best results are naturally to be expected when the disease is strictly confined to one lobe, but we feel nevertheless that when there is advanced disease, say in one lower lobe, with a slight amount in the opposite base or middle lobe of the same side, one is still justified in removing the grossly diseased part. Bronchial carcinoma of the peripheral type with no X-ray or bronchoscopic evidence of mediastinal involvement is an indication for lobectomy, but primary tumours of this type are unfortunately rare. We feel that a chronic lung abscess or multiple abscesses confined to one lobe would probably be best treated by this operation, but have not yet had a suitable case.

Bronchoscopy and Postural Drainage.—When operating on patients who have much sputum there is considerable danger of bronchopneumonia in the opposite side, and this is one of the most potent causes of death in either

one-stage or two-stage lobectomy. Every effort must therefore be made beforehand to reduce the sputum to the minimum. The amount of sputum can be considerably reduced by bronchoscopic aspirations repeated twice weekly over a period of two or three months combined with continuous postural drainage, the patient lying on the abdomen at night and for the greater part of the day with the foot of the bed raised 10 in. In one case we found that the patient was dry for several hours after bronchoscopy and therefore bronchoscoped her immediately before operation, while in several cases we have slipped in a bronchoscope at the end of the operation and aspirated all secretion from the main bronchi.

We believe that bronchoscopy is an essential accompaniment in all chest surgery.

Phrenic Paralysis.—It is an advantage to have the diaphragm paralysed at the time of operation, and the paralysis should only be temporary, because if the remaining lobe is to return to its full and increased function, the diaphragm must be working. The object in paralysing the diaphragm is to reduce movement at the time of operation and so assist the surgeon when dividing adhesions. In all except three of our cases the phrenic nerve was crushed just above the diaphragm after the thorax had been opened. In *Case* 6, on account of the massive adhesions, the nerve was not seen until they had all been divided, and was therefore left intact. When there are many adhesions some time may be wasted in finding the nerve in the chest. We think therefore that in future it will be wisest to crush the nerve in the neck a few days beforehand, as we did in *Case* 9.

Artificial Pneumothorax.—In five of our cases we attempted to obtain a pneumothorax beforehand, but owing to adhesions this is often impossible and failure to obtain a space does not indicate the type of adhesion, nor is it any contra-indication to operation. The idea that a preliminary pneumothorax adjusts the patient for an open thoracotomy we find to be a theoretical rather than a practical consideration. We have therefore abandoned preliminary pneumothorax, and at the time of operation if there are no adhesions we allow the air to enter the pleural cavity slowly through a small opening.

OPERATIVE TECHNIQUE.

Anæsthesia.—There are two essential points in the anæsthesia: (1) After opening the chest, the lung on the affected side must be allowed to collapse and give ample room for manipulation; (2) Quiet respirations, as violent movement of the lung causes a flopping of the mediastinum and shock.

Shenstone and Janes advocate spinal anæsthesia. In our first two cases we employed 'avertin', 0·11 grm. per kilo. of body weight, but this has the disadvantage that the respirations are very shallow—in fact continuous oxygen must be administered while the chest is open—but it has the advantage that one does not require an expert anæsthetist.

For our last nine cases we have given a preliminary injection containing morphine gr. $\frac{1}{6}$, hyoscine gr. $\frac{1}{150}$, and atropine gr. $\frac{1}{100}$, and nitrous-oxide-oxygen during the operation at a very slight positive pressure: the percentage of oxygen varies from 18 to 25; there should be the minimum of re-breathing

as the CO_2 causes increased movement of the lung. This we think is the ideal if one has an anæsthetist with considerable experience and a McKesson's type of apparatus.

For forty-eight hours before operation the patient is given abundant glucose in all drinks and provided with barley sugar to eat. Towards the end of the operation the patient is given 1 litre of 5 per cent glucose at 115° F. per rectum.

Position of Patient.—The patient lies on the good side on a flat table, but if there is much secretion in the trachea the table is tilted to about 15° Trendelenburg position to encourage drainage from the mouth.

Incision.—Our experience so far has been confined to lobectomies for disease in the lower lobe. Although we have exposed the upper lobe on several occasions by this approach we have not yet performed an upper-lobe lobectomy. The technique, however, would be identical except that the chest is opened through the fourth interspace instead of the seventh. In the following description we are considering a lower-lobe lobectomy.

The incision through the skin and extracostal muscles runs in a straight line from the costal margin in front along the seventh interspace to the mid-line behind. All bleeding points in the skin, subcutaneous tissue, and muscle are carefully picked up with artery forceps, and we have found it a considerable saving in time to coagulate these vessels with a diathermy current rather than to tie each one with catgut. The lateral margin of the erector spinæ is then freed, and the posterior ends of the 7th and 8th ribs are exposed. Next the intercostal muscles in the middle of the incision are divided for about 3 in., the space is widened by pulling the ribs apart with small hook retractors, and so the parietal pleura is exposed and incised. If there is a rush of air into the chest, the opening is immediately plugged with the finger and the air allowed to enter slowly during an interval of a minute or two. Having found a pleural space, the intercostal muscles and pleura are then rapidly divided for their entire length with a pair of scissors. At the posterior end this incision keeps near the lower rib so as to avoid the intercostal vessels. The erector spinæ is now retracted, and with a pair of straight bone-cutting forceps the posterior ends of the ribs above and below the incision are divided opposite the tips of the transverse processes without previous elevation of the periosteum. Before the ribs are spread apart pleural adhesions in the neighbourhood must be separated. The fingers of the two hands are then inserted into the chest and the ribs slowly but forcibly pulled apart, for 4 to 5 in. in an ordinary adult. An illuminated rib retractor[11]* is then inserted and opened out to maintain this space.

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* This instrument is a modification of the ordinary abdominal retractor. The arms are longer and slightly curved so that they will lie against the chest wall; also the blades are 2¼ in. wide and curved to a semicircle. In addition it carries a 3-volt lamp on each side for illumination. The light carriers, which are curved to the shape of the blades, clip on to the arms, and at their other ends are connections for the electric leads. These leads are rubber-covered and sterilized by boiling. The lights will illuminate 'round the corner' where the operating-room lights cannot reach, and this is a great assistance when dividing adhesions in the costo-phrenic sinus or apex prior to lobectomy. The lights on the retractor are not sufficient to replace all other illumination during the operation.

Pleural Adhesions.—In lobectomies performed for bronchiectasis we have encountered adhesions in every case: between base and diaphragm only in 2 cases, completely surrounding the lower lobe but not the upper in 5 cases, and with both lobes involved in 2 cases. In *Case* 8, for carcinoma, there were no adhesions.

These adhesions are usually thin and sheet-like and practically avascular, with the exception of those which run from the inferior surface of the lobe to the diaphragm, which are thicker and often contain blood-vessels that require to be ligatured. Early in the series we abandoned a case on account of the extensive adhesions, but since our experience has increased we have done cases just as bad as this; in fact, we doubt if there are any adhesions which cannot be overcome by careful dissection.

All adhesions surrounding the lower lobe must, of course, be separated, and it is advisable to free the costal surface of the upper lobe in order to allow it to expand. The lobes are separated along the oblique fissure, but it is unnecessary to split this right down to the vessels unless there is a natural plane of separation. Finally the pulmonary ligament is divided up as far as the inferior pulmonary vein, and in doing this one may encounter one or two blood-vessels that require to be ligated. A pedicle will now be produced round which the operator's finger and thumb should be able to meet. We have a special pair of lung forceps and long scissors with curved points which we find excellent for dissection and division of adhesions. Care should be taken not to draw up the diaphragm and cut it when dividing basal adhesions; when working in front of the vertebral bodies the extreme proximity of the opposite pleura in this position must be remembered, and in this region the œsophagus on the left and inferior vena cava on the right must be noted and avoided.

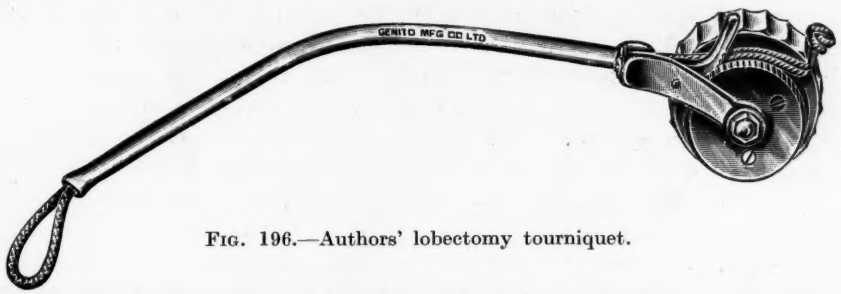

Fig. 196.—Authors' lobectomy tourniquet.

Application of Tourniquets and the Removal of the Lobe.—The loop of a tourniquet* (*Fig.* 196) is slipped over the diseased lobe and kept as high up the pedicle as possible while the assistant winds in the cord and tightens it as much

* The authors' tourniquet consists of a tube which is oval in section, is 9 in. long, and bent in the middle to an angle of 140°. The end that comes against the pedicle is slightly splayed out and has thick lips, while at the other end is a drum on which the cord of the tourniquet is wound. The tube is threaded, from the drum end, with a loop of thick blind cord, the loose ends of which are knotted together and are then attached to the drum by fitting into a groove on the rim. The drum will wind in either direction, but when the ratchet-catch is applied the cord can only be wound up. In practice the catch is not used until most of the loop has been wound in; it is then applied and the cord steadily tightened up round the pedicle.

as he can. A second tourniquet is then employed and tightened up round the lung 1½ in. distal to the first (*Fig.* 197). The pleural cavity is packed off with four or five large dry gauze swabs, which also cover over the margins of the wound in the chest wall. The pedicle is then divided half-way between the cords with a pair of curved scissors. Whilst dividing this the surgeon must constantly note his relation to the proximal tourniquet, and the assistant will find that he can tighten up the cord two or three more points

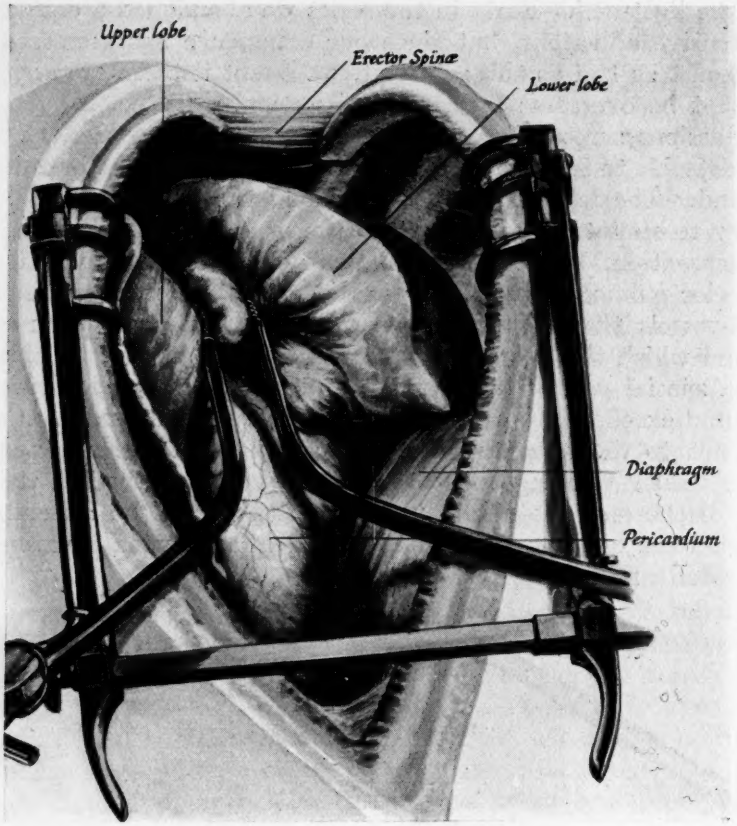

FIG. 197.—The two tourniquets in position.

on the ratchet. Any pus that escapes must be carefully mopped up with small swabs on holders, and when the lobe has been removed the pedicle is swabbed with 1–1000 flavine and then the gauze packs are removed.

Closure of the Pedicle.—The cut end of the pedicle projects for about ½ in. beyond the proximal tourniquet and presents a central white area consisting of blood-vessels and bronchi and a small margin of grey lung tissue (*Fig.* 198). The pedicle is sutured with No. 1 chromicized catgut on a round-bodied half-circle needle. This passes through the white broncho-vascular area as if this was a homogeneous structure and draws it together; the fringe of lung is not included in the first two rows (*Fig.* 199). The suture is continuous; it starts at one end of the pedicle, and after reaching the other

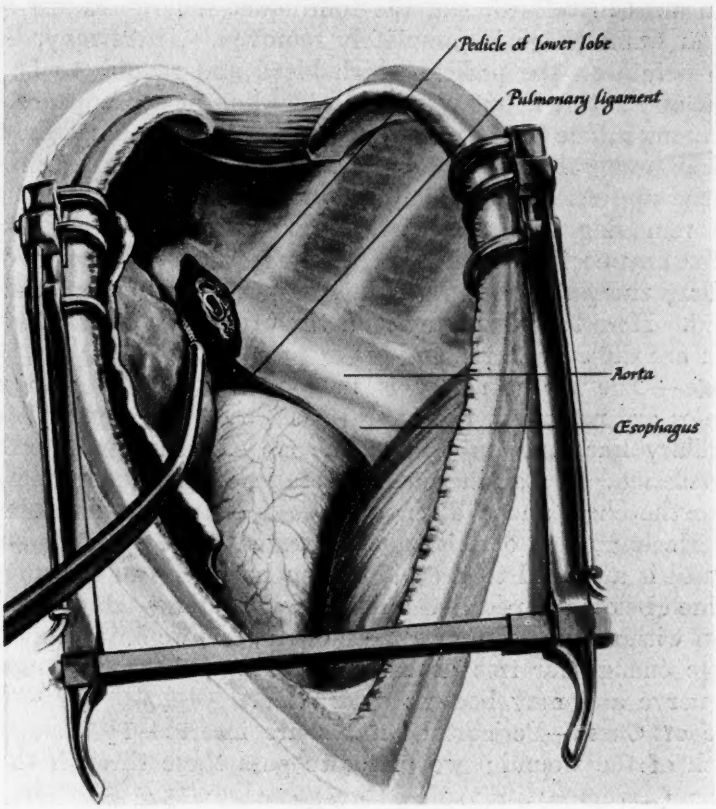

FIG. 198.—The pedicle after removal of the lower lobe.

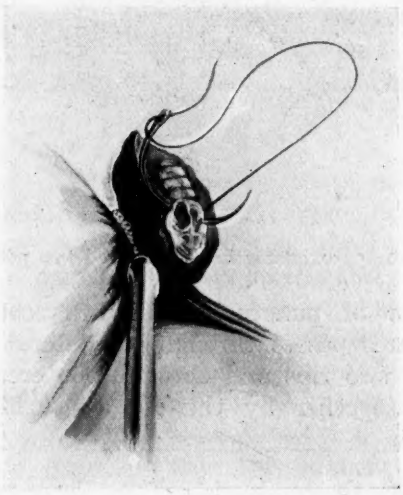

FIG. 199.—The first row of the continuous suture closing the pedicle (semi-diagrammatic).

end returns as a second row to the first point, where it is tied. The vessels and bronchi are thus closed, and the tourniquet is now relaxed a little and if there is no hæmorrhage is completely removed. There may, however, be one or two points on the pedicle which bleed and require to be picked up with forceps and sutured, but usually no bleeding of consequence occurs. A third continuous suture then draws the fringe of lung tissue across the surface of the pedicle, and when this is complete the pedicle is buried into the under surface of the upper lobe by three or four sutures.

Before removing the retractor the pleural cavity must be carefully inspected for any oozing vessels, the most frequent sites being on the diaphragm, along the pulmonary ligament, or posteriorly where the ribs have been divided. Here the diathermy is of the utmost assistance in coagulating areas which are difficult to tie.

Drainage.— The rib retractor is next removed. The skin and extracostal muscles are retracted, the lateral surface of the 9th rib is exposed in the mid-axillary line, and an inch of the rib excised by the ordinary subperiosteal resection. A stab incision is made through the skin and extracostal muscles into the chest and a drainage tube is drawn from within outwards. The intrathoracic portion of this tube has several side openings and is about 4 in. long and is attached to the diaphragm by a loop of catgut so that the tip lies an inch below the pedicle. Shenstone and Janes advocate an ordinary stab incision without resection of a piece of rib, but we have found that if the tube is large enough for free drainage it causes pain from pressure on the intercostal nerve and may become obstructed.

Closure of Chest.—Pericostal sutures are inserted 1½ in. apart for the whole length of the wound; we prefer to pass these through the centre of

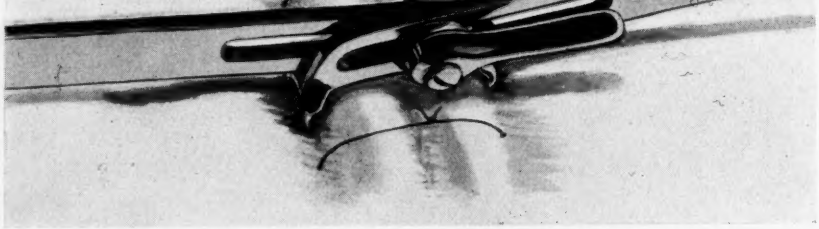

FIG. 200.—The rib approximator in position and two pericostal sutures.

the interspace for fear of puncturing the intercostal vessels. The most posterior of these sutures passes through the lateral margin of the erector spinæ, then round the two ribs and through the erector spinæ again. The ribs are then drawn together by the rib approximator* (*Fig.* 200), the

* The authors' rib approximator consists of two hook retractors which are united together by a bolt that runs in a groove on the shaft of the two retractors: they can be quickly locked or released by a half turn of the bolt.

ligatures tied, and the instrument is removed. The extracostal muscles are sutured with one long continuous suture taking all layers together, and the skin by another continuous suture.

TIME AND SHOCK.

We have found that neither the blood-pressure falls nor the pulse-rate rises when the chest is opened, when the tourniquet is applied, or when the pedicle is divided. The amount of shock is proportional to the extent of the adhesions, because on them depends the length of the operation and the amount of bleeding. For example, there were no adhesions in *Case* 8, and although the man was 55 he left the table with a pulse of 132 and systolic blood-pressure of 120, whereas in *Cases* 6 and 7 there were very extensive adhesions so that the operation lasted nearly two hours in each case, and there was considerable shock at the end.

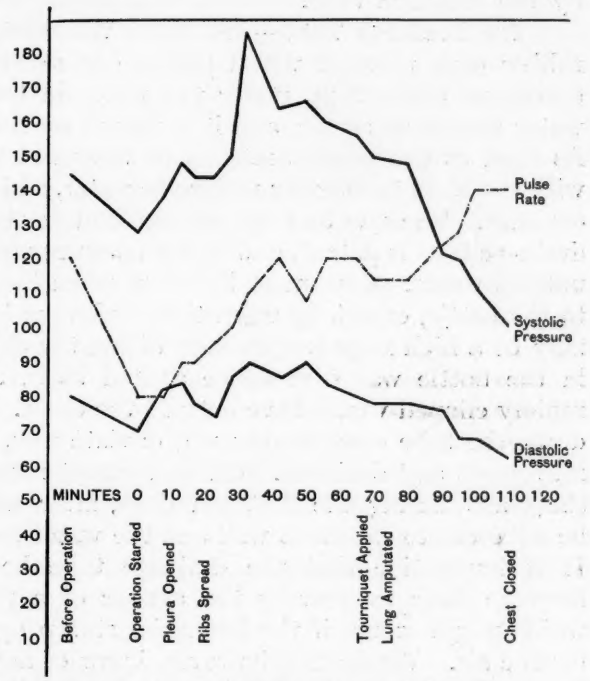

FIG. 201.—*Case* 7. Chart of blood-pressure and pulse-rate during operation.

We agree with Lilienthal that in a major thoracotomy more than in any other operation in surgery time is of the utmost importance. He states that about forty-five minutes is the maximum, but from our experience we find that we can safely take twice as long before the blood-pressure begins to fall and the pulse-rate to increase. The accompanying chart (*Fig.* 201) of blood-pressure and pulse-rate shows a phenomenon that we have observed in most of the cases where these records have been kept, i.e., a rise of blood-pressure during the first hour of operation; and it is not until after an hour to an hour and a half that the blood-pressure begins to fall and the pulse-rate to increase. Towards the end of the operation there is always an improvement when the chest has been closed.

We find that our average time of operation is one and a half hours, the shortest time being one hour and the longest just over two hours. Time may be saved during the course of the operation by the use of a diathermy current for coagulation of blood-vessels, the help of an experienced assistant who can divide from his side the adhesions which the surgeon cannot reach, and the closure of the extracostal muscles and skin by continuous sutures.

Other factors in the prevention of shock are a rigid attention to hæmostasis and a quiet anæsthesia with avoidance of excessive mediastinal movement.

POST-OPERATIVE TREATMENT.

Blood Transfusion.—It is advisable to have the patient grouped before operation and a blood donor available at short notice, and in those cases where the operation has lasted more than one hour we have usually given a transfusion of 300 to 400 c.c.

Position in Bed.—The patient is put in the sitting-up position so that any pleural effusion will gravitate to the bottom of the cavity and be removed by the drainage tube.

The Drainage Tube.—We use a Winchester bottle with a wide neck and rubber cork through which passes one short glass tube and one long one extending down 2 in. under the water in the bottle. This bottle and the water should be sterile, and it is placed on the floor beside the patient's bed. As soon as the patient returns to the ward the drainage tube is connected with the glass tube going under the water, and the air bubbles out as the lung expands. The rise and fall of the fluid in the glass tube indicates that the drainage tube is patent, and if this becomes obstructed it must be manipulated until it works. Care must be taken when the drainage tube is first attached to the bottle, especially when the diaphragm has not been paralysed, as there may be a high negative pressure within the chest. In one such case the fluid in the bottle was seen to be sucked back, and unless the tube had been rapidly clipped would have entered the chest. A screw clamp, which narrows down the tube considerably will obviate this, while still allowing respiratory movement and drainage. On no account must air be allowed to escape into the chest during the first ten days, after which time the upper lobe will be adherent to the chest wall and the water bottle may then be disconnected. It is imperative that the drainage tube be kept patent during the first fourteen days to prevent the danger of a positive pressure pneumothorax which might result if the bronchial stump opened and there was no escape for the air. We do not, however, know of any cases where this complication has occurred in any operations performed by this technique.

POST-OPERATIVE COMPLICATIONS.

Primary Hæmorrhage.—This has occurred in two of our cases. In *Case* 5 the chest had to be opened up the same evening and a small spurting artery was found in the pulmonary ligament and ligatured. As no vessel had been ligatured in this position during operation it must have been temporarily occluded, as the chest was dry before being closed. In *Case* 7 the bleeding was a slight ooze and was controlled by the injection of 20 c.c. of 3 per cent 'coagulin' and the clamping of the drainage tube for twelve hours.

Secondary Hæmorrhage.—This occurred only in *Case* 10. On the fourteenth day after operation a sudden and severe bleeding took place, three pints of blood collecting in the bottle. The tube was clamped and the man given a blood transfusion. On opening the chest a basal empyema cavity

was found which contained blood-clot, but the bleeding had stopped. In view of the rapidity of onset it is probable that the blood had come from the pedicle: it is noteworthy that this is the only case in which the pedicle was not buried in the under surface of the upper lobe at the end of the operation.

Empyema.—Three cases have developed a basal empyema. In *Cases* 6 and 7 this was quite small, but in *Case* 10 the cavity was about 4 in. in diameter. No patient, however, was severely ill from the infection. The empyema has rapidly healed in each case, with the exception of the last one, which is too recent to report finally.

Infection of the Chest Wall.—Infection occurred in three cases. In *Cases* 5 and 10 it followed re-opening of the chest wall for hæmorrhage. *Case* 6 developed a localized abscess in the chest wall which had to be incised, and a sinus was then found leading into the pleural cavity at the posterior end of the wound; after drainage the pleural sinus closed within a week.

Subphrenic Abscess.—Subphrenic abscess occurred in *Case* 10, and was due to injury to the diaphragm at the time of operation.

Bronchopneumonia.—This developed in one patient only (*Case* 5), who had had a severe primary hæmorrhage.

Bronchopleural Fistula.—An obvious blowing fistula has occurred in three cases only (*Cases* 6, 7, and 10). Since our third case we have injected lipiodol into all the sinuses, and in two cases (*Cases* 4 and 8) we discovered a small bronchial fistula which had been entirely unsuspected. As the fistulous track is a long one, it tends to heal rapidly as the drainage tube is gradually shortened.

Cerebral Abscess.—In *Case* 1 the patient died from a suspected frontal abscess ten days after operation, and at autopsy an abscess was found in the left frontal lobe. It was 2 in. in diameter and well localized with thick walls, and on this account the pathologist considered that it must have been present before operation.

REPORTS OF CASES.

Case 1.—Sarah W., aged 13, schoolgirl, admitted to St. Bartholomew's Hospital on May 9, 1932 (referred by Dr. R. Hilton). Her father had died three years previously from pneumonia. There had been nineteen children in the family, and thirteen of these had died from pneumonia.

History.—At the age of 2 the child had diphtheria, for which tracheotomy was necessary, and this was followed by pneumonia; ever since then she had had a chronic cough. At 5 and at 11 years of age there were further attacks of pneumonia. For the past fifteen months the sputum had been becoming more copious, for the past nine months it had been foul, and for five months there had been shortness of breath. Apart from this, the child's general health had been good and her weight had steadily increased.

On Examination.—A red-faced plump child, marked fœtor of the breath, weight 74 lb., no pyrexia, pulse 90, respirations 25. There was an old interstitial keratitis of the right cornea. The heart was displaced to the left, but the sounds were natural. The chest showed poor expansion, most marked at the left base. There was impairment of percussion note at both bases, and diminished breath-sounds. At the left base there was an area of bronchial breathing; there were

many râles, but more on the left than right. In the abdomen neither the spleen nor the liver was palpable. The urine contained no albumin. An X-ray showed that the heart was displaced to the left, and the left diaphragm was slightly higher than normal. The curved outer margin of an atelectatic left lower lobe could be seen through the heart's shadow. Lipiodol demonstrated a severe bronchiectasis in the collapsed lobe, while the rest of the two lungs was healthy (*Fig.* 202).

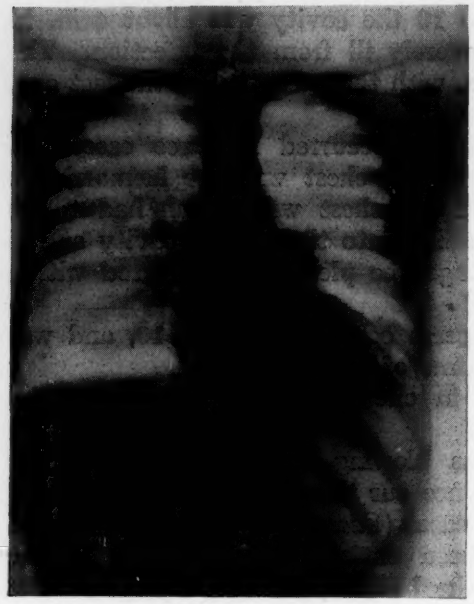

FIG. 202.—*Case* 1. Lipiodol demonstrating congenital bronchiectasis of the left lower lobe.

OPERATION (May 25, 1932). — Left lower-lobe lobectomy. Anæsthetic (by Mr. Rait-Smith), avertin and continuous oxygen after the chest was opened. Incision through the seventh interspace on the left side. The phrenic nerve was crushed where it lay on the pericardium. The left lower lobe presented a marked contrast to the upper lobe : it was diminutive in size and of a dark purplish-grey colour. There were only a few adhesions over the costal surface of this lobe, but firm adhesions to the diaphragm and in the interlobar fissure. These were separated without much difficulty and the lobectomy performed. The chest was drained by the insertion of a tube through a stab incision in the ninth interspace in the posterior axillary line. The operation lasted about one and a half hours.

POST-OPERATIVE COURSE.—The patient had practically no post-operative fever ; the pulse-rate varied from 52 to 110 ; the respirations were at an average of 30 per minute. The child complained of a considerable amount of pain on the left side, which we think was due to the intercostal drainage tube. The general condition was, however, very satisfactory, but on the fourth day after operation the child appeared to be listless and looked as if her head was too heavy. The next day she had severe left frontal headache and was drowsy. There was definite drooping of the left eyelid, but on examination of the nervous system the only abnormal sign to be discovered was an extensor plantar response of the right foot, although this sign did not appear to be constant. On this day the pulse-rate was 110, and the blood-pressure 135/75. The headache persisted and the patient was drowsy at one time and restless at another. No further physical signs developed. A cerebral abscess was suspected, but in the absence of more definite signs craniotomy was considered inadvisable. On the night of the ninth day the child was restless, and then became listless and drowsy, and did not notice the things about her. The right pupil was very dilated and the left eyelid was drooping. Early the next morning the child suddenly started to choke, and pus appeared from the nose and was also coughed up from the mouth, and she died within a few minutes.

POST-MORTEM EXAMINATION.—This revealed a large abscess in the upper part of the left frontal region of the brain, reaching right through to the cortex, with marked flattening of the convolutions. This abscess had a fibrous wall about 3 mm. thick, and from its appearance the pathologist thought it was probably of more than ten days' duration. The examination of the thorax showed that the right pleural cavity was obliterated by adhesions, the underlying lung was healthy and there was no bronchiectasis present. On the left side the lower lobe had been removed. In the region of the pedicle there was a small potential space, but no fluid or sepsis was present. The upper lobe was expanded to fill the rest of the

hemithorax, and its lower surface was adherent to the diaphragm. The lung tissue itself appeared to be quite healthy.

PATHOLOGICAL REPORT.—On section the diseased left lower lobe presented the typical appearance of a congenital atelectatic lobe—namely, a number of thick-walled dilated bronchi surrounded by fibrous tissue, with almost complete absence of any parenchyma (*Fig.* 203).

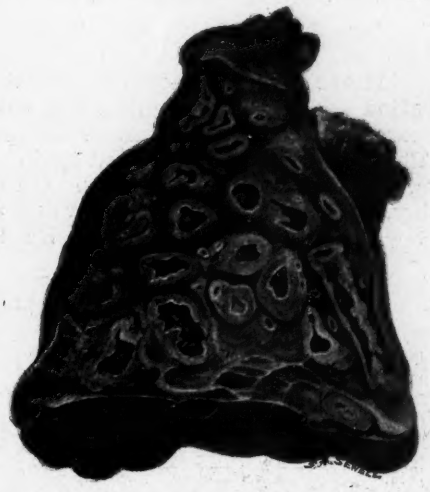

FIG. 203.—*Case* 1. Section across left lower lobe, showing a congenital type of bronchiectasis.

Case 2.—Mary R., aged 23, clerk, admitted to the Brompton Hospital on April 22, 1932 (referred by Dr. Wall).

HISTORY.—Quite well until 1930, when she had a sudden hæmoptysis. Since then she has continued to spit blood in clots at least once a week, and has noticed that it is worse a day or two before menstruation. Coughs only when bleeding. Has lost weight recently. Admitted to the Brompton Hospital in July, 1931, under Dr. Wall.

ON EXAMINATION. — There were no physical signs in her chest; her heart was normal; blood-pressure 120/90. X-ray on July 31 showed that the heart and mediastinum were displaced to the left but no infiltration of the lungs. After the injection of lipiodol and a more heavily exposed film a dense opaque area could be seen through the cardiac shadow and the lipiodol failed to enter the left lower bronchus. The patient was therefore referred to one of us (J. E. H. R.) for bronchoscopy. This demonstrated that the left lower-lobe bronchus was completely blocked by a very vascular growth, which bled profusely when a piece was removed for section. The section of the biopsy specimen showed chronic inflammatory tissue only. Further bronchoscopies performed a month later demonstrated a stenosis at the entrance to the left lower lobe. The patient was therefore bronchoscoped on seven further occasions and the stricture was dilated with bougies and granulation tissue painted with silver nitrate. In spite of this treatment the patient still continued to spit up blood. Therefore exploratory thoracotomy was advised with a view to lobectomy. On May 17 an artificial pneumothorax was induced on the left side.

OPERATION (May 26, 1932).—Left lower-lobe lobectomy. Anæsthetic (by Mr. Hewer), avertin and nitrous-oxide-oxygen. Incision through the seventh intercostal space; the phrenic nerve was crushed just above the diaphragm; the left lower lobe was small and airless and of a firm consistency; there were a moderate amount of adhesions surrounding this lobe; it was impossible to use the distal tourniquet owing to the consistency of the lobe. At the end of the operation the chest was drained by a stab incision in the ninth interspace.

POST-OPERATIVE COURSE.—This was uneventful except for some intercostal pain owing to the drainage tube. This was removed on June 6. The patient left hospital completely healed on July 10. The patient was seen in the Out-patient Department on April 28, 1933. She stated that during the last three months she had had four hæmoptyses. They all occurred at night when she was in bed, and she attributed them to nose-bleeding. Also since leaving hospital she had lost 6 lb. in weight. She had no cough or sputum, and no pain except slightly during damp weather. On examination she looked thin but healthy; there was a good air entry at her right base. Under the X-ray the diaphragm was high, the movements were normal but limited in extent; the heart was displaced to the left, and there was some pleural thickening in the left lower zone, but apart from this no abnormal shadow could be seen.

PATHOLOGICAL REPORT.—On section the diseased lobe showed that the main lower-lobe bronchus was dilated to about an inch in diameter and projecting into it was a bright yellow necrotic looking growth. The rest of the lung tissue was collapsed. The microscopic section showed that the growth was undoubtedly malignant and probably a squamous-celled carcinoma of undifferentiated type.

Case 3.—Annie R., aged 43, housewife, admitted to the Brompton Hospital on April 26, 1932 (referred by Dr. Bosanquet).

HISTORY.—Pneumonia in childhood. Eight years previously was under observation in hospital for pulmonary tuberculosis. Has had a cough off and on for years. Three years previously had pleurisy, but does not remember which side. In April, 1932, the cough started again and she had pain in the left side of her chest and several small hæmoptyses. On admission she had 2 oz. of purulent sputum a day, with dyspnœa on exertion and pain in the left side of her chest.

ON EXAMINATION.—A thin but healthy-looking woman, weight 115 lb. Heart slightly displaced to the left, but sounds normal. The lungs gave an impaired note at the left base, with bronchial breathing and many coarse râles. Abdomen normal. Sputum was negative for tubercle bacilli. X rays showed the heart displaced to the left, fibrosis of the left base, and a suggestion of bronchial dilatation in the left lower lobe. This was confirmed by lipiodol (*Fig.* 204). There was no evidence of dilatation in any other lobes of the lung. The patient was under observation in hospital for three months, during which time she gained weight and the sputum decreased to 2 drachms daily. An artificial pneumothorax was induced on July 18 and refilled every other day.

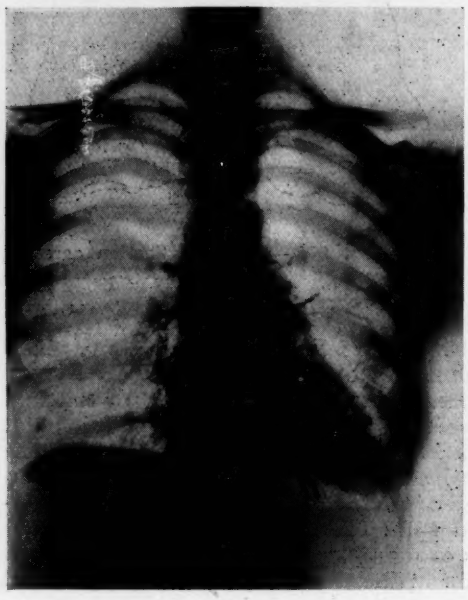

FIG. 204.—*Case* 3. Lipiodol demonstrating acquired bronchiectasis of the left lower lobe.

OPERATION (July 28, 1932, at the Centenary Meeting of the B.M.A.).—Left lower-lobe lobectomy. Anæsthetic (by Mr. Hewer), nitrous-oxide-oxygen. Incision through the sixth interspace. The phrenic nerve was crushed just above the diaphragm. The lower lobe was of normal size and appearance when compared with the upper lobe. There were no adhesions except for a few between the lower lobe and the diaphragm and in the interlobar fissure, which made the operation extremely simple. The chest was drained through a stab incision in the eighth interspace in the mid-axillary line.

POST-OPERATIVE COURSE.—This was entirely uneventful. The tube was removed on the fourteenth day, and from this day the patient began to get up. She left hospital completely healed on the twenty-eighth day after operation. In December, 1932, she was seen and was well with no cough or sputum. On May 28, 1933, the patient stated that her general health was excellent, and this winter was the first she had gone through without having a cold. She had no cough, but said she occasionally brought up sputum in the morning; no hæmoptyses, but occasional pain in the chest during damp weather. On examination the chest was symmetrical and moved equally on the two sides; a good air entry was noted and no added sounds. Under the X-ray the left diaphragm was seen to be raised and fair movement was present on inspiration. The heart was displaced to the left, and there was some fibrosis at the left base.

PATHOLOGICAL REPORT.—On section the diseased lobe presented the typical appearance of an acquired bronchiectasis in the lower and medial part of the lobe

—namely, very little peribronchial fibrosis, six to eight dilated bronchi with thick walls, while the rest of the lung showed normal healthy-looking parenchymal tissue. An illustration of this type is shown in *Fig.* 209.

Case 4.—Charlotte S., aged 32, housewife, admitted to the Brompton Hospital on July 25, 1932 (referred by Dr. Burrell).

History.—Left-sided pneumonia in November, 1931, followed by cough and purulent offensive sputum, which has persisted. In December, 1931, there was an hæmoptysis of one pint. On admission she still had the cough and a trace of sputum which was slightly blood-stained. There were also occasional pains in the left side of the chest.

On Examination.—A well-grown thin woman of a good colour, weight 111 lb. No clubbing of fingers. Heart normal; lungs showed poor movement and impaired percussion note at the left base behind, with bronchial breathing over this area, increased voice conduction, whispering pectoriloquy, and a few coarse râles. X rays

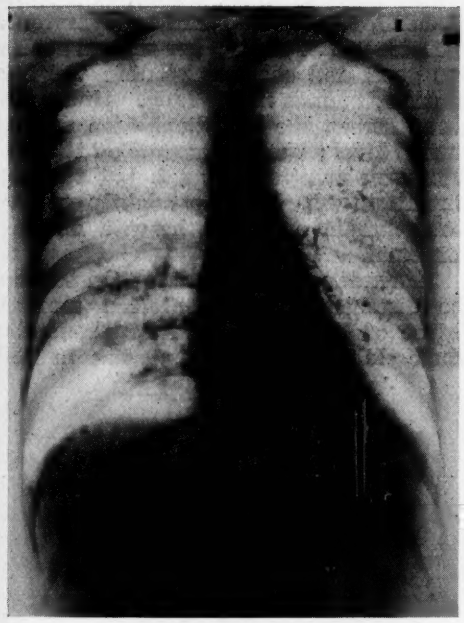

Fig. 205.—*Case* 4. Lipiodol demonstrating advanced bronchiectasis of the left lower lobe.

Fig. 206.—*Case* 4. Lateral view.

showed the heart slightly displaced to the left, fibrosis at the left base with suggestion of bronchial dilatation. This was confirmed by lipiodol, which showed a very marked basal bronchiectasis involving the left lower lobe only (*Figs.* 205, 206). Pre-operative artificial pneumothorax was attempted but not obtained.

Operation (Aug. 27, 1932).—Left lower-lobe lobectomy. Anæsthetic (by Mr. John Hunter), nitrous-oxide-oxygen. Chest opened through the sixth interspace. There were many thin adhesions over the lower lobe and a few over the upper. These were all divided. The phrenic nerve was then found and crushed just above the diaphragm. The lower lobe was firm and airless, smaller and of a darker colour than the upper lobe. At the end of the operation the chest was drained through a stab incision in the seventh interspace in the mid-axillary line. The operation lasted about one and a half hours and the condition at the end was very good.

Post-operative Course.—On Sept. 17 the patient had been running a temperature of 100° every evening since the operation. Therefore an X-ray was taken on this day, which showed a small fluid level at the base. The tube was

therefore removed and replaced by a catheter. Following this the temperature returned to normal. On Sept. 27 lipiodol was injected into the sinus and demonstrated a long track running upwards towards the root of the lung and a small unsuspected bronchial fistula. The patient was discharged two days later with a small tube. The tube was removed a week later and the sinus promptly healed. On Jan. 6, 1933, the patient was seen again. She was very well and had no symptoms. There was a poor air entry at the left base. X rays showed nothing abnormal except a rather high diaphragm with very limited but normal movement (*Fig.* 207). On April 28, 1933, the patient had no symptoms referable to her chest; her general health was good and she was gaining weight; no added sounds in the lung. Under the X-ray screen the left diaphragm was of normal level; the movements on the two sides were practically equal, but the left diaphragm was held up slightly in the costo-phrenic angle. Otherwise nothing abnormal.

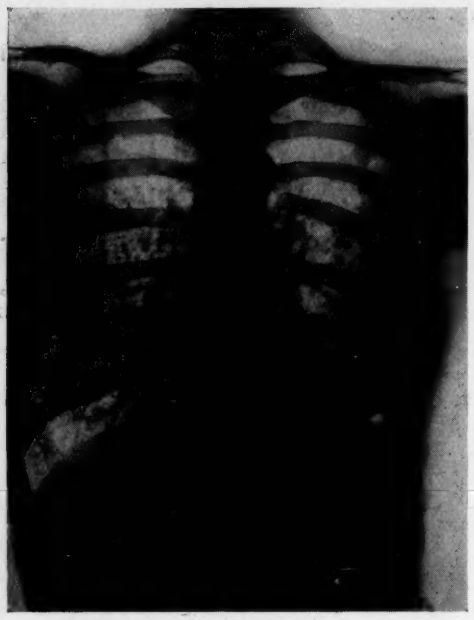

FIG. 207.—*Case* 4. X-ray of the chest four and a half months after left lower-lobe lobectomy.

PATHOLOGICAL REPORT.—On section the diseased lobe presented a marked degree of bronchiectasis throughout the whole lobe, with pneumonitis in the remaining pulmonary parenchyma (*Fig.* 208).

FIG. 208.—*Case* 4. Section of the left lower lobe showing a marked degree of bronchiectasis with pneumonitis.

Case 5.—Ernest W., aged 19, clerk, admitted to St. Bartholomew's Hospital on Oct. 8, 1932 (referred by Dr. Geoffrey Bourne).

HISTORY.—Twelve years previously he had had pneumonia; then quite well until five years ago, when he developed a cough with a little sputum; this did not bother him, and his general health remained good until one year ago, when he coughed up about half a cupful of blood. Since then has had a nervous breakdown, the cough became worse, the sputum increased, and he lost weight.

ON EXAMINATION.—A tall youth, thin but healthy-looking, weight 142 lb. Slight clubbing of nails. The chest moved equally, with a slight flattening on the left side; left base percussion note impaired, air entry less than the right; harsh breath-sounds with a few added sounds. X rays without lipiodol showed nothing abnormal,

but after introduction of the oil an area of bronchiectasis was seen in the left lower lobe of moderate extent. The right side and left upper lobe were normal. A pre-operative artificial pneumothorax was induced on Oct. 18 and a moderate collapse obtained.

Operation (Oct. 26, 1932).—Left lower-lobe lobectomy. Anæsthetic (by Mr. Frankis Evans), nitrous-oxide-oxygen. Incision through the seventh interspace. The phrenic nerve was crushed just above the diaphragm. The left lower lobe did not present any contrast in size or appearance from the upper. There were some adhesions surrounding the lower lobe, while the upper lobe was free. The operation was performed by the usual technique, but instead of draining the chest through a stab incision in this case, we resected 1 in. of the 10th rib in the posterior axillary line, as described in the text. The operation lasted sixty-five minutes and at the end the pulse-rate was 90 and the blood-pressure 98/52.

Fig. 209.—*Case* 5. Section of the left lower lobe showing an acquired type of bronchiectasis.

Post-operative Course.—Four hours after operation we were notified that the pulse-rate had been steadily increasing and was now 140 and there was a little over a pint of blood in the drainage bottle. It was obvious that the patient was bleeding, and it was decided to re-open the chest. A transfusion of 800 c.c. of blood was given and the patient anæsthetized with gas and oxygen. The whole incision was re-opened and a small spurting artery could then be seen at the lower end of the pulmonary ligament. It was clamped and tied. No vessel in this position had been tied during the operation so it must have been temporarily occluded. The patient developed a post-operative bronchial pneumonia at the right base and coughed up about 6 oz. of greenish sputum daily for the first two weeks, after which time he gradually got better. He also had considerable pain in the left side of the chest, and on the right side from pleurisy. He was out of bed thirty-five days after operation and left hospital on Jan. 4, 1933, at which time he had no cough or sputum, the temperature, pulse, and respirations were normal, and he said that the 'tickling sensation' in his chest had completely gone.

On May 28, 1933, the patient had no symptoms whatsoever, was gaining weight, and feeling well. He said, " It is a real boon to me to be able to go about in company and no longer feel embarrassed, as I used to before." Under the X-ray the left diaphragm was seen to be raised, but slight normal movement was present; the heart was displaced to the left, but apart from this nothing abnormal was seen.

Pathological Report.—On section the diseased lobe presented the typical appearance of an acquired bronchiectasis, and, as shown in *Fig.* 209, the upper half, i.e., that portion supplied by the dorsal bronchus, was absolutely normal, while the lower half contained the dilated bronchi and some degree of pneumonitis in the parenchyma.

Case 6.—Henry O., aged 39, warehouseman, admitted to the Brompton Hospital on Oct. 10, 1932 (referred by Dr. Thomas Nelson).

HISTORY.—Twenty-nine years previously he had had pleurisy, but does not remember which side. In August, 1931, he had twenty-three teeth extracted under a general anæsthetic, and shortly after that developed a cough, which has continued. In May, 1932, he started to have an offensive sputum, which was most noticeable in the morning or on exertion. In June had to stop work owing to pain in the right side of the chest, increase of the cough and sputum, increasing shortness of breath, and loss of weight.

ON EXAMINATION.—A thin, ill-looking man, weighing only 91 lb. He had no pyrexia. The heart was displaced towards the right; on the right side there was poor movement, impaired percussion note, weak breath-sounds, bronchial breathing, and râles both in front and behind. The rest of the physical examination was normal, and the urine contained no albumin. X rays showed movement of the right diaphragm restricted, mediastinal structures displaced to the right, left lung normal, while the right lung showed partial collapse of the lower lobe, with fibrosis and suggestion of bronchiectasis. This observation was confirmed by lipiodol, which also demonstrated some dilatation in the terminal branches of the right middle lobe. On Oct. 25 a pre-operative artificial pneumothorax was attempted, but only a small pocket could be obtained.

OPERATION (Oct. 29, 1932).—Right lower-lobe and partial middle-lobe lobectomy. Anæsthetic (Mr. Hewer), nitrous-oxide-oxygen. Incision through the seventh right interspace. Very dense and widespread adhesions throughout the hemithorax, but most marked over the middle and lower lobes. The phrenic nerve could not be found. The right lower lobe was first removed and the pedicle completely sutured. The lower margin of the middle lobe presented many hard nodular areas, but as there was no fissure between the upper and middle lobes, a middle-lobe lobectomy by the usual technique was impossible, and it was therefore decided to do a partial pneumectomy of the middle lobe. Two intestinal clamps were placed across the middle lobe and the distal two-thirds excised. There was considerable bleeding at the hilum region where the ends of the two clamps met, and artery forceps had to be applied. The cut surfaces of the lung were oversewn and the hilum closed by a continuous catgut suture. (This partial amputation of the middle lobe was very unsatisfactory and we decided never to employ it again. We consider it would have been better to have created a fissure between the upper and middle lobes by cutting into the lung with a diathermy knife and then using the tourniquets as in a lower-lobe lobectomy.) The chest was closed and drained by resection of a portion of the 9th rib. The operation lasted just over two hours, and by the time the chest was closed the patient's pulse was only just palpable at the wrist. He was therefore given a blood transfusion and intravenous saline before leaving the operating table.

POST-OPERATIVE COURSE.—The patient ran a temperature of 101·5° and developed a discharge from the upper end of his incision, and at the end of ten days there was a purulent discharge from the drainage tube. On Nov. 26 it was found that the discharging sinus at the upper end of the wound communicated with a subcutaneous abscess and also went down to the cut ends of the two ribs, which could be felt to be necrosed. This portion of the wound was laid open and the subcutaneous abscess drained dependently. Following this the patient's temperature returned to normal. On Dec 16 he had a small quantity of purulent discharge from the chest, there was an obvious bronchial fistula, and examination with a probe (confirmed by X rays) showed that there was no cavity but a small track inside the chest which led up to the fistulous opening: there was a small sinus at the upper end of the wound going down to the necrosed rib. On Jan. 10, 1933, the patient was up; the sinus at the upper end of the wound had healed; there was still a tube in the chest wall and a bronchial fistula, which, however, sounded much smaller.

On Jan. 26 another patient in the ward developed influenza, and our patient caught it. His temperature suddenly rose and he had fine crepitations in the left lower lobe. On Feb. 2 he died with influenzal bronchopneumonia.

POST-MORTEM EXAMINATION.—This showed that the right side of the chest was occupied by the upper lobe and the pleura was universally adherent. The remaining lobe showed old healed tubercle at the apex, some œdema but no pneumonia. On the left side the upper lobe showed healed tubercle at the apex, but otherwise normal. The left lower lobe showed bronchopneumonia in the stage of red hepatization.

PATHOLOGICAL REPORT.—On section the diseased lobes presented an appearance very similar in type to that found in *Case* 4—namely, a severe degree of bronchiectasis with much pneumonitis in the surrounding lung tissue.

Case 7.—Robert M., aged 24, optical worker, admitted to the Brompton Hospital on Oct. 26, 1932.

HISTORY.—Eight years previously he had suffered from sudden hæmoptysis which lasted for two weeks; following this he was quite well and returned to work. Had occasional cough and slight sputum, but not sufficient to bother him. Five years ago he had another hæmoptysis similar to the first. In September, 1932, he had his third hæmoptysis, which cleared up after a few days, and he felt quite well; no cough, pain, or shortness of breath.

ON EXAMINATION.—A healthy-looking, well-covered man: heart normal, blood-pressure 160/76. The lungs showed slight impairment of percussion note at the left base, with bronchial breathing and bronchophony, but no added sounds. The white blood-count was 8700. The X rays showed that the heart and mediastinum were displaced considerably to the left and that the left base was opaque so that the diaphragm could not be distinguished. On introduction of lipiodol large pools of the oil collected at the base, which suggested that the oil was in the stomach, but it was eventually decided that it was in the lung (*Fig.* 210).

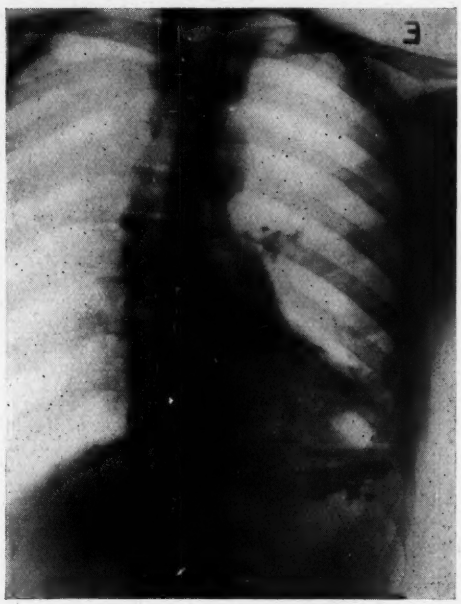

FIG. 210.—*Case* 7. Lipiodol demonstrating large cystic cavities in the left lower lobe. Patient standing.

OPERATION (Nov. 5, 1932). — Left lower-lobe lobectomy. Anæsthetic (Mr. John Hunter), nitrous - oxide - oxygen. Incision through the eighth interspace. There were a very large number of short strong adhesions throughout the hemithorax. The lower lobe was dissected out with extreme difficulty. The phrenic nerve was found when sufficient adhesions had been divided and was crushed just above the diaphragm. There was troublesome bleeding from the loose cellular tissue of the mediastinum in the region of the œsophagus which was eventually stopped by hot packs. At the end of the operation a drainage tube was inserted by the resection of one inch of the 9th rib in the mid-axillary line. The operation lasted two hours, but the blood-pressure did not fall nor the pulse-rate rise until the last half hour. At the end of the operation the pulse was 140 and the systolic pressure below 80.

POST-OPERATIVE COURSE.—The patient was returned to the ward and given an immediate transfusion, following which the pulse-rate fell to 120 and the blood-pressure rose to 85. Six hours later the pulse-rate had risen to 124 and there was a definite trickle of blood coming from the drainage tube in the chest: 20 c.c. of 3 per cent 'coagulin' were introduced through the drainage tube, which was then clamped. Before this was done it was noted that the breath-sounds were strongly audible over the left upper zone, which suggested that the lung had already expanded. During the next ten days there was a blood-stained discharge from the chest. On

Dec. 1, as the patient had been running an evening temperature of 100° to 101°, an X-ray was taken which showed that the drainage tube was kinked and there was a small collection of fluid. The tube was adjusted and the temperature fell to normal. On Dec. 9 he had an obvious bronchial fistula, with air bubbling out of the drainage tube on coughing. The tube was therefore disconnected from the bottle and he started to get up. Lipiodol on Dec. 14 showed that there was a small basal empyema with fistula. On Jan. 10, 1933, there was a small sinus in the chest wall and a fistula: practically no discharge; the patient was up and well; no hæmoptysis.

When seen on April 28 the patient stated that his fistula had healed three weeks previously. He had a sinus in his chest wall about 2 in. long; the small drainage tube,

FIG. 211.—*Case* 7. Section of the left lower lobe showing a congenital cystic type of bronchiectasis.

therefore, was removed. He had no symptoms. Under the X-ray the diaphragm was seen to be raised, and had a slight paradoxical movement. The heart was displaced to the left, but otherwise there was nothing abnormal. On May 5 the sinus was completely healed.

PATHOLOGICAL REPORT.—On section the diseased lobe contained two very large cysts and several smaller ones (*Fig.* 211). Microscopic section showed that the epithelium was similar to that found in the trachea, with long cilia.

Case 8.—George K., aged 55, bus driver, admitted to the Brompton Hospital on Sept. 14, 1932 (referred by Dr. R. A. Young).

HISTORY.—For many years he had had slight cough with expectoration in the morning. Three years previously, i.e., in 1929, he first spat up a little blood, and since then this had occurred on many occasions. In May, 1932, he was treated for hæmorrhoids by injection. Since then he had noticed that he had been losing weight amounting to 28 lb., and had had general weakness and malaise. On admission he had a dry cough, no sputum, but pain in the right chest.

ON EXAMINATION.—A large, rather thin-looking man and in no distress; weight 164 lb.; no clubbing. His chest was emphysematous; heart not displaced; arteries somewhat thickened and tortuous; blood-pressure 130/80. On the right side in the lower part of the axilla there was an area of slightly impaired percussion note, with weak breath-sounds and fine râles; otherwise the chest was normal. Apart from this, nothing abnormal was discovered on general examination, and the urine was normal. Sputum was negative for tubercle. Blood-count normal. X-ray

examination showed mediastinum and diaphragm normal: in the lower zone on the right side there was an irregular rounded opacity which extended out to the chest wall but was not continuous with the mediastinum (*Fig.* 212). In a lateral view it was shown to be just posterior to the heart shadow and with a peaking of the diaphragm opposite it. Lipiodol failed to enter this area. Bronchoscopy on Oct. 15 showed no evidence of enlargement of the mediastinal lymphatic glands and nothing abnormal was discovered. While under observation in hospital the patient started to have purulent sputum up to 1 oz. daily. He had a temperature of between 99° and 100° on most evenings and a leucocytosis of 15,700. The diagnosis rested between a chronic lung abscess or growth, but on account of these findings it was considered that the former was the more probable.

Operation (Nov. 5, 1932).—Operation for first stage of lung abscess under local anæsthesia. Five inches of the 7th and 8th ribs were resected in the mid-axillary line, and when resecting the intercostal bundle between these two the pleura was accidentally torn. The lung was then seen to be completely free and the pleural opening was therefore enlarged and the lower lobe palpated. On the outer surface of this lobe was a white scarred area about 1½ in. in diameter, and on palpation a hard spherical lump about 3 in. in diameter could be felt in the lower lobe. The diagnosis between chronic abscess and carcinoma was still in doubt, but it was decided that lobectomy could be very rapidly performed and would be the wisest course. Therefore the patient was given nitrous-oxide-oxygen anæsthesia by Mr. Scadding (the Assistant Medical Officer), the incision was enlarged forwards and backwards, and a *right lower-lobe lobectomy* performed. In cutting across the pedicle no enlarged lymphatic glands were discovered. During the first half of the operation the patient's blood-pressure steadily rose, the operation was completed in one hour, and when the patient left the table the systolic blood-pressure was 120 and the pulse only 132.

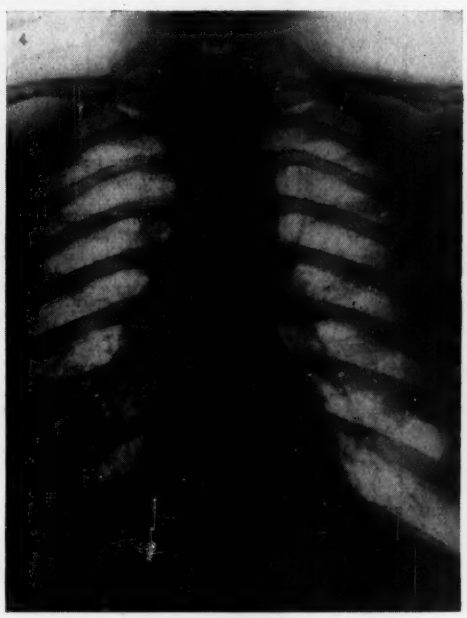

Fig. 212.—*Case* 8. X-ray of the chest showing an irregular rounded opacity in the lower zone of the right lung.

Post-operative Course.—In spite of this man's age he had a more satisfactory post-operative course than any of the other cases. Six hours after operation his pulse-rate was only 80 and strong. His temperature and pulse chart is shown in *Fig.* 213. On Dec. 17 the tracheobronchial lymphatic glands were irradiated by the insertion of fifteen radon seeds of 1·5 mc. each by the method described by one of us (H. P. N.[12]). There was no reaction following this, and the patient left hospital on Dec. 23 with no cough and the wound healed.

On April 28, 1933, the patient stated that he was "feeling fine" and had gained two stone in weight since leaving hospital; he had no symptoms at all. Under the X-ray the right diaphragm was seen to be raised, and some movement was present on inspiration; the aorta was markedly dilated; in the right lung there was some thickening of the pleura and a shadow obscuring the right costo-phrenic angle. Fourteen radon seeds were seen in the mediastinum. The oblique view showed an indefinite opacity towards the posterior part of the right lung, which may represent consolidation breaking down in the centre.

Pathological Report.—On section the diseased lobe showed the appearance seen in *Fig.* 214. Microscopically this proved to be a squamous-celled carcinoma.

Case 9.—Jessie H., aged 19, machinist, was admitted to St. Bartholomew's Hospital on Nov. 26, 1932.

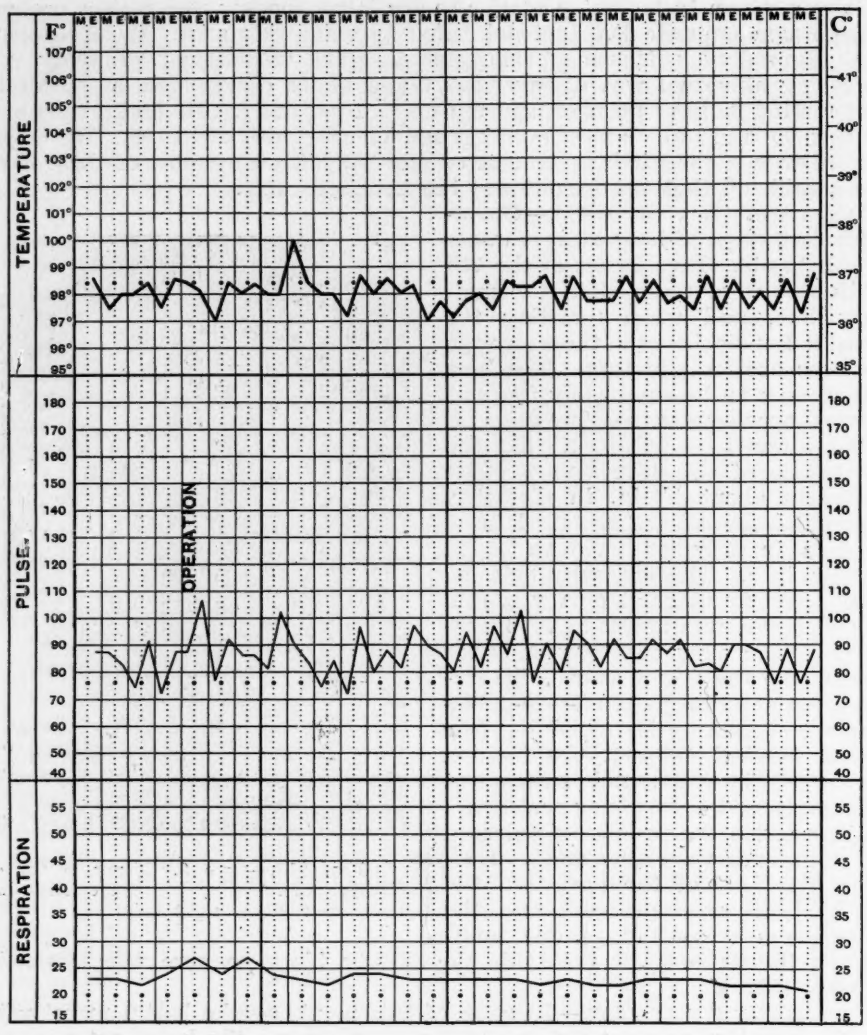

FIG. 213.—*Case* 8. Chart of the temperature, pulse, and respiration.

HISTORY.—There was a history of a cough and sputum for as long as she could remember. In January, 1932, she was admitted to the hospital for acute appendicitis, and following operation attention was drawn to her chest by the cough and copious sputum. There were signs of bronchiectasis at the left base, and lipiodol demonstrated a moderately severe degree of cylindrical dilatation in the left lower lobe. Nine months later she was readmitted to hospital as she still had 1 or 2 oz. of sputum daily, which was of foul taste and smell. On Nov. 28 the left phrenic nerve was exposed in the neck and crushed.

OPERATION (Nov. 30, 1932).—Left lower-lobe lobectomy. Anæsthetic (by Mr. Frankis Evans), nitrous-oxide-oxygen. Incision through the seventh interspace. The lower lobe was slightly smaller than normal, but the usual colour. There were adhesions surrounding the lower lobe, but only one or two over the costal

surface of the upper lobe. At the end of the operation the chest was drained by resection of a portion of the 9th rib. The operation lasted for exactly one hour. The blood-pressure at the beginning of the operation was 126/84 and the pulse-rate 84, while at the end of the operation the blood-pressure was 116/74 and pulse-rate 112.

FIG. 214.—*Case* 8. Section of the right lower lobe showing a squamous-celled carcinoma.

POST-OPERATIVE COURSE.—This was entirely uneventful. The closed drainage tube was disconnected on the fourteenth day, and the patient left hospital on Jan. 1, 1933, with normal temperature, pulse, and respirations, chest healed, and no sputum. On Jan. 6 the patient was feeling well and had no cough or sputum. On March 17 she was back at work; she had a slight cough, with ½ oz. of sputum in the twenty-four hours. On examination of her chest there was poor movement at the left base but no added sounds could be heard in the lung. On April 7 she had a cough with only a trace of sputum, and was feeling quite well. On May 12 lipiodol filling demonstrated some bronchiectasis in the lower portion of the left upper lobe, which accounted for the persistence of sputum.

PATHOLOGICAL REPORT.—On section the diseased lobe presented an appearance almost identical with that seen in Case 5 (*see Fig.* 209).

Case 10.—Thomas S., aged 38, tram conductor, admitted to the Brompton Hospital on Nov. 17, 1932.

HISTORY.—He was quite well until July, 1926, when he coughed up a small clot of blood and was in bed for eight days with pain on the right side of the chest.

In December, 1927, he had several hæmoptyses of about 4 oz. followed by one of 20 oz. He spent ten weeks in hospital at this time and then had another severe hæmoptysis. From March, 1928, for two years he attended the Out-patient Department at one of the London chest hospitals, having frequent small hæmoptyses, and he was diagnosed as bronchiectasis. In 1932 he had six very severe hæmoptyses. Since 1928 he had had a small quantity—less than 1 oz—of mucopurulent sputum. He had always felt well in himself.

On Examination.—He was a tall, well-grown man, obviously very anæmic. General examination revealed nothing abnormal except at the right base behind, where there was a slightly impaired note and weak breath-sounds, with some post-tussic râles. The blood examination showed red blood-cells 4,500,000, hæmoglobin 66 per cent, white blood-cells 5000; the differential count was normal. X rays without lipiodol showed apparent bronchial dilatation at the right base; the left lung field appeared normal. After lipiodol, which unfortunately had entered the alveoli, some dilatation of the branches of the right descending bronchus could be seen. On Dec. 2 bronchoscopy showed some dilatation of the right main and middle bronchi. Although the cause of the hæmoptysis was still unknown, we considered that the seat of the trouble lay in the right lower lobe, and it was therefore decided to remove this lobe on account of the very severe bleeding. A pre-operative artificial pneumothorax was attempted but not obtained.

Operation (Dec. 10, 1932).—Right lower-lobe lobectomy. Anæsthetic (by Mr. John Hunter), nitrous-oxide-oxygen. There were a large number of firm adhesions over the lower lobe and a very well-marked fissure partly separating the apex of the lower lobe (the dorsal 'lobe') from the rest. After suturing the pedicle the end appeared to be so satisfactorily sealed with the lung tissue that the stump was not buried in the upper lobe on this occasion for the first time. The duration of the operation was one and a half hours, and at the end the condition was fair, but on return to the ward the patient was given a blood transfusion.

Post-operative Course.—The patient's condition was on the whole satisfactory, but he ran a temperature up to 103° most evenings. The discharge from the chest soon became purulent, although the wound itself looked satisfactory. On Dec. 19 it was noted that he had 12 oz. of pus daily from the tube and was coughing up 1 oz. of sputum: the temperature was up to 102°. On Dec. 25 the patient suddenly collapsed, and it was found that the drainage bottle contained about one pint of blood. The drainage tube was immediately clamped and the patient was given a transfusion, and under nitrous-oxide-oxygen anæsthesia the chest was rapidly opened up. A basal empyema was discovered about 4 in. in diameter, some blood-clot was found in the cavity, and the end of the stump appeared necrotic with two bronchial fistulæ. There was a small vessel on the chest wall that was bleeding, but it was considered that this was probably the result of re-opening the chest and that the hæmorrhage had really come from the pedicle, although at the time the chest was opened (two hours after the bleeding) it had stopped. The cavity was packed with dry gauze and the wound left open.

When considering the cause of this man's empyema and secondary hæmorrhage we realized that this was the only case in which this calamity had occurred and was also the only occasion on which we had not buried the bronchial stump in the under surface of the upper lobe.

The infection in the wound gradually cleared up, and it became smaller, but in spite of this the patient's temperature continued. He complained of pain, and there was tenderness over the costal surface of his liver. An X-ray suggested a subphrenic abscess, which was confirmed by aspiration. Following the drainage of this abscess, the patient's temperature returned to normal. We consider that this subdiaphragmatic abscess resulted from injury to the diaphragm when dividing basal adhesions at the time of the lobectomy.

On March 18, 1933, the subphrenic abscess had healed. The patient started to get up, and was feeling very well. On April 7 he was discharged home to attend as an out-patient twice weekly. The chest wound was reduced to a sinus 2½ in. deep and about 1 in. in diameter, with two minute bronchial fistulæ at the bottom

of the wound. There was practically no discharge from the wound; the patient had no cough, and there had been no recurrence of his hæmoptysis since the lobectomy. On May 12 the fistula and sinus were completely healed.

PATHOLOGICAL REPORT.—On section, the diseased lobe presented an appearance which we had not seen in any of the previous cases. One of the large tertiary bronchi was dilated in a saccular manner to the size of a primary bronchus: the wall of this dilatation was about 2 mm. thick and contained cartilage. The section of lung supplied by this bronchus was hard and fibrous, and contained many minute dilatations. The rest of the lower lobe, i.e., about nine-tenths of it, presented an entirely normal appearance.

SUMMARY OF CASES.

1. The *ages* of our cases varied from 13 to 55. Five cases were females and five males. The *predominant symptom* in six cases was cough and foul sputum, and in the other four a repeated hæmoptysis.

2. The *disease* for which lobectomy was performed was in two cases primary bronchial carcinoma and in eight cases bronchiectasis. Extensive pleural *adhesions* were present in seven cases, and practically none in three cases. *Lobes removed*: in seven cases the left lower, in two the right lower, and in one the right lower and middle lobes.

3. *Post-operative complications*: hæmorrhage in three cases, basal empyema in three cases, bronchopneumonia in one case, bronchial fistula in five cases (two of these were silent), and cerebral abscess in one case. The *average length of time* from operation to healing of the chest-wall sinus was five weeks.

4. *Present condition* (May 12, 1933):—

Dead (from cerebral abscess ten days after operation) ..	1 case
Dead (from influenzal pneumonia three months after operation)	1 ,,
Healed and free from symptoms	6 cases
Back at work, but with slight symptoms	2 ,,

CONCLUSIONS.

Although this series of ten cases is too short to justify the expression of results by percentages, it appears that by this technique the mortality of lobectomy may be considerably reduced, and that this operation will take its place as the treatment of choice in cases of unilobular bronchiectasis.

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BRONCHIECTASIS:

A STUDY OF THE PATHOLOGY OF SIXTEEN SURGICAL LOBECTOMIES FOR BRONCHIECTASIS.

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THE problem of bronchiectasis, its etiology, pathology, and treatment, has held the interest and attention of clinician and pathologist alike for many years. Laennec[1] (1826) was probably the first to refer to this condition. The next important contribution to the subject was that of Corrigan[2] (1838), who, in a paper on cirrhosis of the lung, reported four cases. He considered the disease to be primarily a fibrosis of the lung with dilatation of the bronchi produced by traction of the scar tissue.

ETIOLOGY.

Congenital Factors.—As most of the early reported cases were noted in children, it was natural that a congenital factor should be emphasized as important in the etiology of this disease. This was first suggested by Grawitz[3] (1880) when he reported a series of cases, mostly in children, all of which he believed were congenital in origin. Barlow,[4] in the same year, reported a case in a child of 3 months. In the interior of the upper lobe of the left lung he found a cyst the size of a Spanish chestnut containing air, but with no apparent opening into it. Sauerbruch,[5] who is probably the greatest exponent of this theory, believes that 8 per cent of the cases are congenital in origin. A congenital predisposition was thought by Duken[6] to be important in some cases. Hedblom[7] suggested the possibility of the development of the disease in a persistent fœtal atelectasis when the chest cavity later expanded beyond the capacity of the atelectatic lung.

Mechanical Factors.—Mechanical factors, such as stenosis of the bronchi from foreign bodies in the lumina or from external pressure by aneurysm, enlarged glands, tumour growth, etc., have been emphasized by a number of writers (Meyer,[8] Schott,[9] Lemon,[10] Hedblom,[7] Biermer,[11] and Brunn and Faulkner[12]). It has, however, been generally admitted by these men that an added infection of the bronchial wall was a necessary concomitant. Such a mechanical factor as increased intrabronchial air-pressure in coughing is given by Schneider[13] as sufficient cause in itself to produce one type of bronchiectasis, viz., the atrophic type. Biermer[11] thinks the mechanical pressure of retained secretions in the lumen added to an increased intrabronchial air-pressure is important.

Mechanical stresses such as lateral pulls on the walls of the bronchi from fibrosis of the lung parenchyma, pleural adhesions, collapse of the lung, etc., are mentioned as causes in some cases, (Jex Blake,[14] Coplin,[15] Coats,[16] Brooks,[17] Hedblom,[7] McFarland,[18] and Kaufmann[19]). Hedblom says that atelectasis in adults may produce bronchiectasis by virtue of an increased negative pleural pressure, lessening as it does the support to the outside of the bronchial wall and allowing the atmospheric pressure to produce its dilating effect. More recently Warner,[38] studying bronchiectasis from a clinical and radiological standpoint, laid stress upon the mechanical influence of increased negative intrathoracic pressure occurring in cases of lobar atelectasis. He has shown that bronchiectasis may develop within a few weeks of the onset of a massive collapse or atelectasis. Infection, however, he believes, precedes these changes, producing a weakening of the walls of the larger bronchi and a swelling of the mucosa with obstruction of the bronchioles.

Infection.—Infection of the bronchial wall is generally accepted by all as being of most importance. Graham and Warner,[39] in their study of bronchiectasis associated with triangular shadows, conclude that "Infection is the all-important factor". Its relation to chronic sinus disease is emphasized by Lemon,[10] Webb,[20] and Adam,[21] the sinuses being the primary source of infection. A chronic infective process in the wall of the bronchus, however, is considered by others as being a complication or sequela of such diseases as measles, whooping-cough, bronchopneumonia, and acute bronchitis (Schneider,[13] Lemon,[10] Jex Blake,[14] Hedblom,[7] Klare and Reusse,[22] McNeil et al.,[23] and Findlay and Graham[24]). The infection, Meyer[8] thinks, is probably due to a loss of antiseptic properties of the bronchial mucus. The retention of copious secretions is given also as a factor by Meyer and by Biermer.[11] Specific types of infection are not mentioned by these authors. Smith,[25] however, from the observations of many clinical cases and the study of much experimental material, concludes that primary bronchiectasis is due to a specific type of infection, namely, fuso-spirochætal infection.

Other Factors.—Other factors have been suggested, such as loss of nerve control of the bronchial musculature (Lebert[28] and Biermer[11]), overwork of the bronchi (Black[29]), and nutritional disturbances from an endarteritis of the bronchial arteries (Brunn[30]). In reference to the latter, Sauerbruch and Brunn did some experimental work on the tying of the branches of the pulmonary artery. They were able thereby to produce a fibrosis the contraction of which caused bronchiectatic dilatations of the bronchi of the affected lobe. Duken[6] and Ochsner[26] believed that in the earlier phases bronchiectasis may be the result of a loss of tone in the muscular wall and the inability of the bronchus to empty itself of secretions.

ANATOMY AND PHYSIOLOGY.

Anatomy.— The bronchial walls vary slightly in structure from the bifurcation of the trachea to their terminations in the lung parenchyma. Throughout their course they are lined by a ciliated type of columnar epithelium supported by a stroma with a number of mucous glands opening on the surface. Macklin[31] has shown that the elastic elements are distributed

in longitudinal as well as circular bundles. There are several layers, the most conspicuous being situated in the mucosa and running longitudinally. They are branched and grouped in fasciculi, allowing thus for dilatation of the bronchi as well as lengthening. Just outside of the longitudinal elastic membrane of the mucosa is another layer running mainly circularly. These latter fibres are interwoven with the muscle bundles. Other layers of lesser importance are also present.

The bronchial musculature extends throughout the whole length of the air-passages (Baltisberger[32]). In the main branches, where the cartilage plates are distributed around the entire circumference of the wall, the muscle fibres form a complete layer located between them and the mucosa. The muscle bundles, while in the main encircling the tube, branch off diagonally to form a more or less continuous network in 'lazy-tong' fashion, or, as Miller[33] expressed it, run in " geodesic lines ". The walls of the main branches down to the point where they have a diameter of 0·5 mm. are reinforced by cartilage plates, which prevent them from collapsing on inspiration.

Physiology.— Macklin[31] has shown that the bronchi during inspiration both widen and elongate. During expiration they contract and shorten. Whether this is done simultaneously throughout the whole tubular system or passes as a wave from larynx to periphery of lung and vice versa is not known. He suggests, however, that a contraction of the bronchi passing along as a wave from periphery to hilus would obviously best serve in emptying the lung of air.

These points in reference to the anatomical structure and physiology of the bronchial system serve to show that it is not a rigid system of tubes operating passively as conduits for the passage of air between the larynx and lung alveoli, but a very actively functioning part of the respiratory system, dilating and lengthening during inspiration to accommodate its many branches to the inrush of air and the expansion of the lung, and then conversely contracting and shortening probably in a wave-like peristaltic movement during the process of expiration. During shallow breathing, of course, this is slight in amount, but becomes considerable with coughing or other violent respiratory movements. The ability of the bronchi to return to a normal diameter depends, as in blood-vessels, upon the integrity of the muscle and elastic elements. Any damage to these structures must of necessity lead to a loss of resiliency and contractile power in the unduly dilated wall. It is this failure to return to the normal state of rest which eventually leads to the permanent state of relaxation and dilatation of bronchiectasis.

PATHOLOGY.

Bronchiectasis is considered as a permanent dilatation of one or more bronchi. Temporary dilatations which have been demonstrated radiologically no doubt are due to weakness and loss of tone of the bronchial muscles continuing over a relatively short space of time. This probably occurs in the early stages of the development of the disease, and if no disintegrative changes take place in the bronchial wall, complete restoration is quite possible. Later with irreparable damage to the essential structures of the wall, particularly

the musculature and elastic elements, a return of the bronchi to their normal diameter is impossible, and dilatations become inexorably fixed. The etiological factors leading to this latter condition may be numerous, but the essential pathological lesion, I believe, is the same in all cases—namely, a weakening or loss of integrity of the musculo-elastic system.

Within the last two years we have had the opportunity of studying, both bacteriologically and pathologically, a series of sixteen cases of bronchiectatic lesions in lobes of lungs freshly removed surgically for intractable symptoms of bronchiectasis. These were removed from patients of both sexes, with ages varying from 14 years to 52 years. A clinical report of this series of cases is presented by Dr. Janes on pp. 273–5. A previous communication by Shenstone and Janes[37] covers in part this same series. The lobe of lung when removed by the surgeon was immediately brought to the laboratory in sterile towels, and here with sterile instruments the affected bronchi were opened and cultures of various types made. The bacteriological findings have been reported in another paper by Dr. Greey.[35] He found in a careful study of nine of the cases that there was no uniform bacterial flora. Spirochætes were found in four of the cases, and pure cultures of streptococcus in three of them.

Fig. 215.—Bronchiectatic lung removed from a female, aged 21 years, with a history of cough since childhood.

The lung specimens were then trimmed and prepared for mounting. Blocks were taken from various areas along the course of the dilated bronchi. These were placed immediately in Zenker's fluid or in 10 per cent formalin. Transverse and longitudinal sections were made, and stained with hæmatoxylin and eosin, Van Gieson, Mallory's phosphotungstic acid, hæmatoxylin, and Masson's stain, and a combination of Masson's and Weigert's elastic tissue stain. This latter combination gave very excellent results. Preparations with Gram-Weigert's stain for bacteria and Levaditi's silver stain for spirochætes were also made.

The important feature of this series is the fact that the material was obtained in a fresh state and in some cases fairly early in the development of the disease. An opportunity was thus afforded to study the evolution of the disease process from the preliminary inflammatory reaction to the final fully developed permanently dilated bronchial tube. The lobes of lung removed at operation were all lower ones, including in some cases a portion of the upper lobe, and from both right and left lungs. The dilatations were of the fusiform or cylindrical types and varied from 4 mm. to 1·5 cm. in diameter (*Fig.* 215). The secondary branches were most frequently involved. The mucous membrane was of a peculiar pinkish-yellow colour, and was soft,

succulent, almost redundant in appearance. The lumina for the most part contained in their proximal portions very little secretion. This was either yellowish and mucoid in character or yellowish-grey and inspissated. The finer bronchioles leading off from the dilated proximal portions were, however, usually well filled with a yellowish mucoid material. The thickness of the walls varied, as a rule being definitely thicker than normal. In the vast majority of them there was little gross evidence of involvement of neighbouring lung tissue beyond a variable amount of lung collapse. In none was there any evidence of tuberculosis.

Histopathology.—An analysis of the histological preparations revealed findings that were fairly constant throughout. A definite inflammation of the wall was found in all cases, with degenerative changes in certain essential

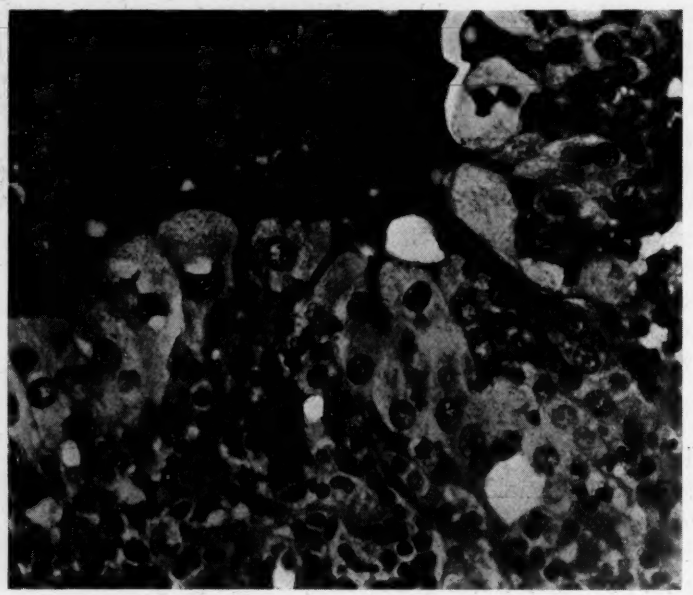

FIG. 216.—Lining cells are swollen, irregular in size and shape, and cilia are for the most part gone. (× 400.)

structures added thereto. The reaction was, however, essentially an inflammatory one. In the more acute cases the beginnings of the process were apparent. The more advanced processes were seen in the longer-standing cases. The more acute and early cases showed a marked hyperplastic state of the mucosal epithelium. The cells were large, swollen, irregular in outline and size, and tended to grow up in a polypoid manner, increasing thereby the thickness of the mucosa. In all but two, however (acute cases), the cilia of the lining epithelial cells were intact. In these two cases the lining cells were swollen, irregular in size and shape, and the cilia for the most part gone. This is illustrated in *Fig.* 216, a case with a three and a half months' history. Considerable quantities of exudate, consisting of mucus with large numbers of polymorphonuclear leucocytes, occluded the lumina of the smaller branches. In view of the frequency of hæmoptysis in these cases it was

interesting to note that very few ulcers of mucosa were found. These occurred only in the more acute cases and were microscopic in size.

The most striking changes, however, were found in the subepithelial layer. Here, lying between the lining epithelium and the cartilage plates,

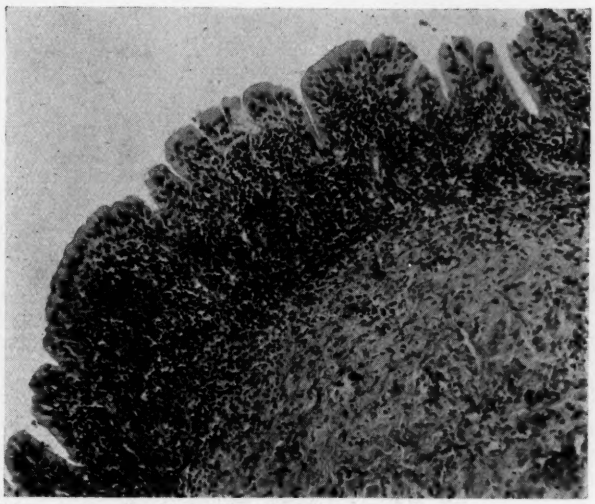

FIG. 217.—Inflammatory infiltration of the subepithelial layer. (× 80.)

are found important structures—namely, the bronchial musculature and elastic fibres. In this zone a most marked inflammatory reaction occurred (*Fig.* 217). The inflammation was somewhat granulomatous in character, the growth

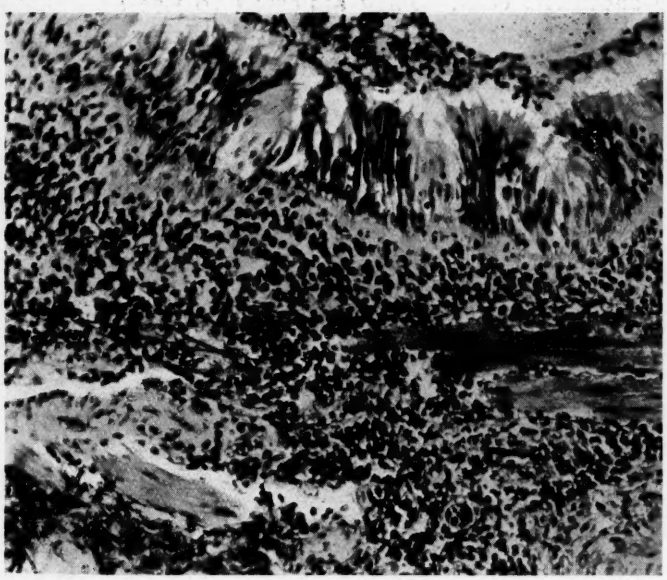

FIG. 218.—Degenerative changes in muscle fibres and some splitting of the elastic lamellæ are shown. (× 160.)

of new blood-vessels and connective tissue being a prominent part of the process. In the earlier stages, as seen in the acute cases, this granulomatous feature was not so apparent, and the muscle and elastic fibres were found for the most part intact. Here and there, however, beginnings of a degenerative

change were seen in the muscle fibres and some sp'itting of the elastic lamellæ was also evident (*Fig.* 218). A moderate infiltration of lymphocytes and plasma cells permeated these tissues and some œdema was present. One frequently saw evidences of weakening of the wall in the outpouching of the

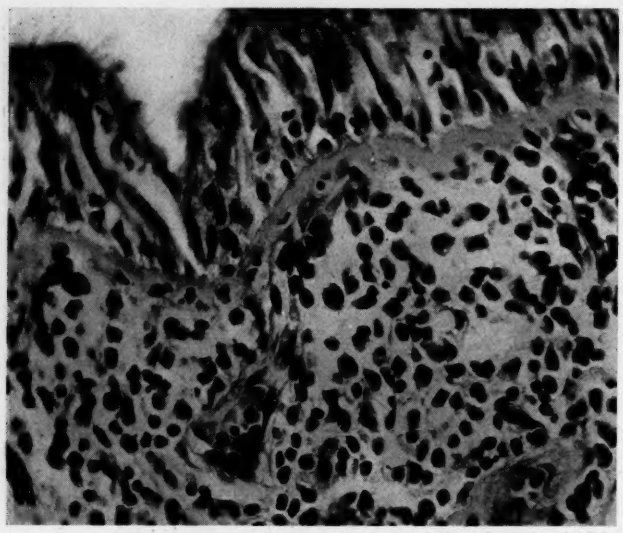

FIG. 219. — Section of mucosa showing character of the subepithelial inflammatory reaction. (× 320.)

mucosa at the points where the muscle and elastic tissues were destroyed. The capillaries of the mucosa stood out prominently, the endothelial layer being made up of large succulent cells. They appeared to rise abruptly in the outer layers and to run at right angles directly to the lining epithelium. In this stage very little increase of connective tissue stroma was seen.

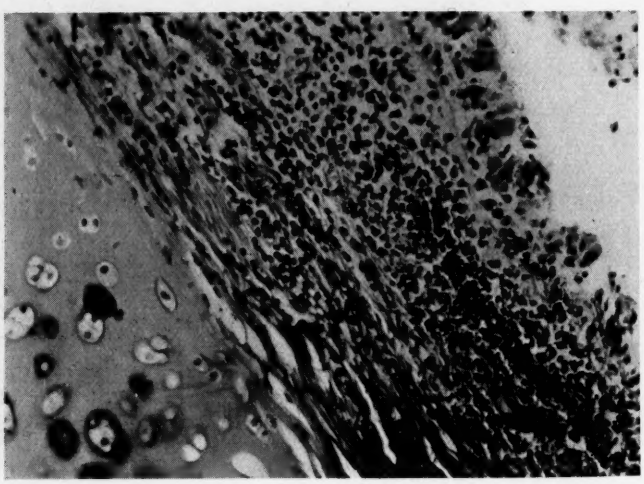

FIG. 220. — Muscle and elastic elements replaced by fibrous or granulation tissue. (× 160.)

In the longer-standing cases no particular change in the character of the inflammatory infiltration was apparent (*Fig.* 219). Destruction of muscle and elastic elements, however, was quite pronounced, and in many bronchi was complete. They were replaced by a fibrous or granulation tissue (*Fig.* 220)

and densely infiltrated with lymphocytes and plasma cells. The capillaries stood out prominently, being lined by one or more layers of large swollen endothelial cells. A peribronchial inflammation became apparent, with some

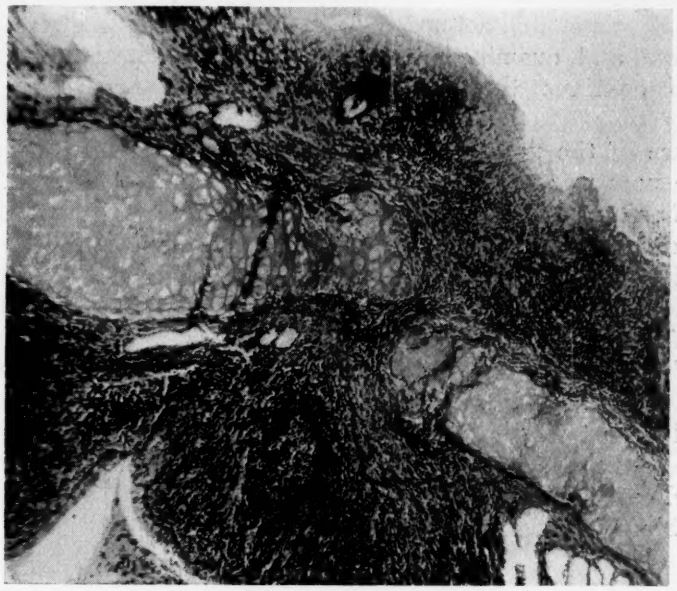

FIG. 221.—Involvement of cartilage plates leading to destruction and replacement by fibrous tissue. (× 48.)

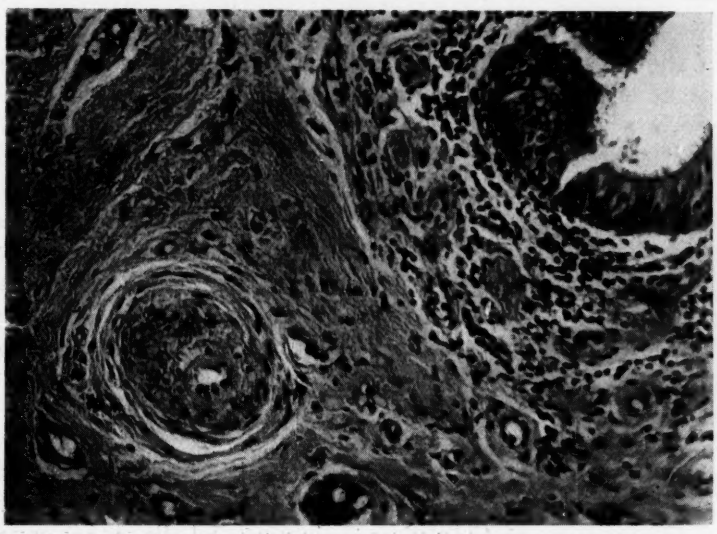

FIG. 222.—Intimal thickening of wall of bronchial artery producing the endarteritis obliterans type of arteriosclerosis. (× 160.)

fibrosis of a narrow zone of the adjacent lung parenchyma. The involvement of the cartilage plates at times was rather extensive, leading to destruction and replacement by fibrous tissue. This is illustrated in *Fig.* 221, where the

process has involved the septa between two bronchi, tending to a melting down of the intervening wall. In the rest of the cases, where the process was more definitely established, it was interesting to note that the lining epithelium was intact throughout, and consisted of normal stratified columnar epithelial cells with well-defined cilia. Fresh scrapings were made from some of these and examined with dark-field illumination. The cilia were observed to continue their whipping movements for some hours after removal.

A feature of the process that perhaps has not been sufficiently emphasized in the past was the involvement of the bronchial arteries. A definite intimal thickening with stenosis of the bronchial arteries was found in 65 per cent of the cases. These lesions were found only in the bronchial arteries and only in those of the dilated bronchi. The thickenings of the walls were intimal and therefore of the type of endarteritis obliterans. This is illustrated in *Fig.* 222. Whether or not this was consequent upon the inflammation of the bronchial wall, or an etiological factor, is difficult to determine While it may not have been of prime importance, it is quite possible that it later played a part in diminishing the blood-supply and lessening the resistance of the tissues. An example somewhat akin to this is seen in the experimental work of Hepler[36] in the production of solitary cysts of the kidney by the combined interference with circulation and obstruction to the urinary outflow.

COMMENT.

The outstanding and most consistent pathological finding in our series was a chronic inflammatory condition of the bronchial walls with various degrees of damage up to complete destruction of the musculo-elastic tissue. Infection of the wall would therefore seem to be of prime importance in the development of this disease. In the early stages the condition is probably of the nature of a paresis of the muscular system leading to loss of tone with consequent dilatation. Provided the infection is of short duration, recovery at this stage might be complete. When, however, the infection has persisted for a longer period of time permanent damage occurs, the resiliency of the wall is lost, and the dilatations become fixed. With the dilatation, even if temporary, a vicious cycle is established: Bronchial secretions tend to accumulate, become infected, with spread to the adjacent walls. The damaged walls then slowly give way under the intermittent strain of ordinary or violent respiratory movements. There was nothing found in our series of cases to indicate that mechanical overstrain such as pleural adhesions, collapsed lung, etc., had played a part.

Mechanical obstructions such as an aspirated foreign body, tumour growth, etc., are found in certain cases. None of our cases, however, revealed such conditions. Where present they play their part, I believe, by causing a local lowering of resistance of mucosa and blocking of normal secretions. Infection then becomes established within the lumen of the bronchus, and from here soon spreads to the wall, involving the whole bronchial tree distal to the obstruction. Here it persists and leads to degenerative changes in the musculo-elastic elements of the wall.

The vascular changes occurring in the bronchial arteries, if not a prime factor in the development of this disease, are no doubt an important contributory one, in that they lower the resistance of the tissues and favour persistence of the infection.

In cases where no mechanical obstruction is present such a thing as a physiological block to the clearing out of secretions might conceivably occur. A persistent local infection at the orifice of one branch may damage the ciliated epithelium to such an extent that the cilia become paralysed and the mucus in the distal portion accumulates to pathological proportions. A metaplasia of epithelium to a squamous-celled type at this point would have the same

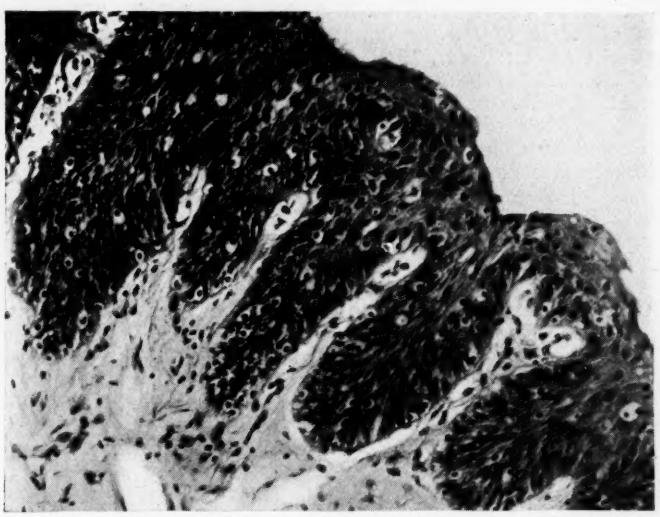

FIG. 223.—Metaplasia of bronchial mucosa from columnar to squamous-cell type. (× 160.)

effect. *Fig.* 223 illustrates this latter type of block. The section was taken from the proximal end of the bronchiectatic tube. In a more recent case, not included in this series, I found all the dilated bronchi in the affected lobe to be lined with squamous epithelium. In such cases there must be a definite physiological block to the clearing of secretions from the more distal portions of the bronchial tree. Following the stagnation of secretions, and infection, a spread through the bronchial wall would lead to the same chain of events and terminate in bronchiectatic dilatations consequent upon a damage to the muscle and elastic elements.

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ETIOLOGY AND TREATMENT OF SLIPPED EPIPHYSIS OF THE HEAD OF THE FEMUR.*

By E. N. WARDLE, Liverpool.

It is proposed to divide this paper into two sections: (1) A consideration of the etiology of the condition; (2) A description of a new method of treatment, with detailed reports of three consecutive cases in which it has been used successfully; followed by a critical survey of the methods employed by other writers, and their recorded results, leading up to the basis on which the present method was evolved.

The clinical findings are well recognized and accurately described in various writings quoted. *Case* 9 of this series was included because it has some bearing on the question of etiology; but it also serves to emphasize one clinical point which is not stressed in the literature—namely, that the signs in this condition are those of a fractured neck of the femur.

The X-ray pictures and their significance are a subject on which there seems to be agreement in general, but a further consideration of this is included later.

ETIOLOGY OF SLIPPED EPIPHYSIS.

First of all a summary of the cases examined, from the etiological standpoint.

Case 1.—J. R., male, aged 15 years. First seen on July 2, 1931, with a history that a month earlier he had fallen off a bicycle and *struck the left knee.* A week after this he began to have pain in the left hip and was unable to bear weight on the limb. Overweight and overheight for his age. (For details, *see* account in second section.)

Case 2.—H. Ma., male, aged 13 years. First seen on April 23, 1931, complaining of a limp. No pain and no history of trauma was elicited. No signs of endocrine disturbance. (*Fig.* 224.)

Case 3.—C. R. W. H., male, aged 14 years. First seen on Nov. 22, 1931.

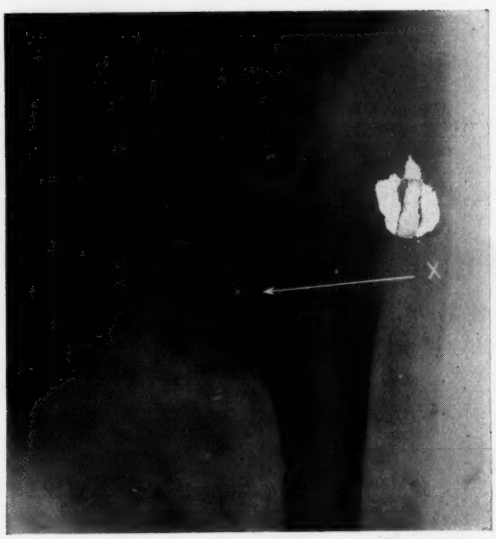

Fig. 224.—*Case* 2. X-ray showing (X) areas of rarefaction in metaphysis.

* A preliminary article on work undertaken for the Lady Jones Research Fellowship in the University of Liverpool.

Had noticed a little pain in the hip one month previously. One week before had *knocked left ankle* while going upstairs, and the day afterwards could not walk on the left leg. (For details, *see* second section.)

Case 4.—H. Mo., male, aged 7 years. No history available.

FIG. 225.—*Case* 5. Right hip. Epiphysis displaced and rarefaction of metaphysis still apparent.

FIG. 226.—*Case* 5. Knees, showing metaphysial expansion and rarefaction in both epiphysis and metaphysis.

Case 5.—W. P., male, aged 16 years. First treated for genu valgum in January, 1930, but reported back to hospital in September, 1931, with 'scissor-leg' deformity, and X rays at this time showed generalized epiphysial involvement, including slipping of one upper femoral epiphysis. (*Figs.* 225–230.)

FIG. 227.—*Case* 5. Involvement of lower epiphyses of tibia and fibula.

FIG. 228.—*Case* 5. Metaphysial expansion and rarefaction in wrists.

FIG. 229.—*Case* 5. Left hip. Marked rarefaction in metaphysis, but displacement of epiphysis is only just commencing.

Case 6.—M. S., female, aged ? years (since dead). X rays show generalized epiphysial involvement, and a diagnosis of renal rickets was made.

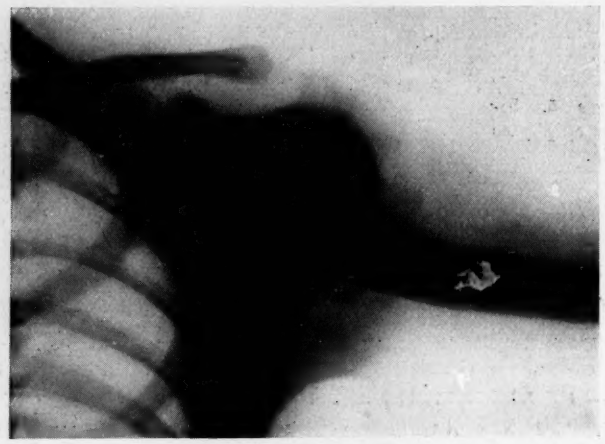

FIG. 230.—*Case* 5. Changes in upper end of right humerus similar to those already shown in right femur.

Case 7.—S. J., male, aged 17 years. History of a fall in September, 1931, in which the right thigh was wounded by a spike. This healed. In November he

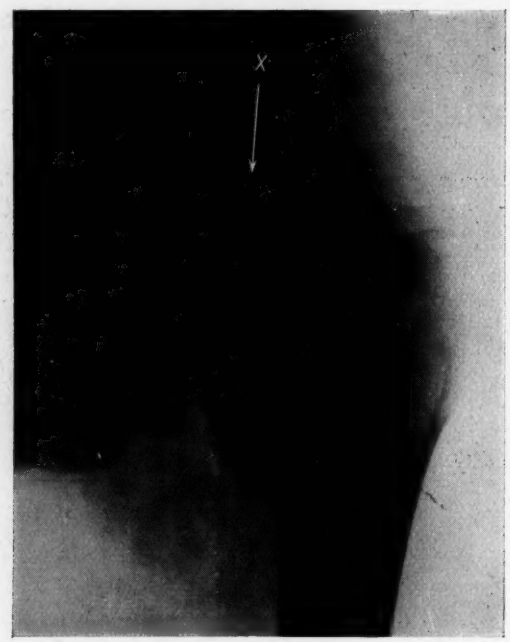

FIG. 231.—*Case* 7. X shows large area of rarefaction in metaphysis.

started to have pain in the right thigh and hip, and by December was unable to bear weight on the limb. (*Fig.* 231.)

Case 8.—R. D., female, aged 4 years. Complete separation of epiphysis of the head of the femur by direct crushing injury. Epiphysis extruded from out of the acetabulum. (*Fig.* 232.)

FIG. 232.—*Case* 8. X shows outline of capital epiphysis, extruded from the acetabulum on to the dorsum ilii. Note that the relation of the greater trochanter to the neck remains unchanged.

Case 9.—J. D., male, aged 13 years. Following an injury to the hip, *a direct blow on greater trochanter*, he was found to lie flexed, adducted, and everted at the hip-joint and to have ½ in. real shortening. A diagnosis of slipped epiphysis was considered, but X rays showed a fracture through the trochanteric region (*Fig.* 233).

FIG. 233.—*Case* 9. Showing fracture through the trochanteric region.

Case 10.—D. L., female, aged 15 years. Four years previously this patient had had a trans-trochanteric osteotomy, and X rays suggest a slipped epiphysis. It was interesting to note that on Jan. 13, 1932, she had 1 in. real shortening, ¼ in. apparent shortening, and that she could abduct 45° at the hip-joint; she had half normal range of rotation, a compensatory left dorsal scoliosis, and walked well without a noticeable limp.

Case 11.—A. G. H., male, aged 17 years. First seen on April 13, 1933. He had noticed that he was walking with a limp for eight weeks previously and that his left knee and ankle 'gave way' easily. There was no history of any previous accident or illness. (For details *see* second section.)

According to the literature there is no general agreement on the question of etiology. While the factors are agreed upon, their relative value is not. From 1907, when Elmslie[4] wrote upon the subject, until 1926 there had been no notable additions to the literature. In that year Key,[1] in an article which contained a considerable amount of original work, also included an excellent résumé of previous writings, and summed up the position with regard to

etiology. He maintained that there were three important etiological factors: (1) Trauma; (2) Static forces; (3) Endocrine disturbance; and that while it could be shown that each of these played a part he was unable to assess their relative value, and stated that the three of them were never present together. Other writers since then have either followed Key or evaded the vexed question of etiology altogether.

Badgley[2] (1929) emphasizes Key's statement that the periosteum around the neck of the femur becomes thinner during adolescence and that the capital epiphysis is thereby deprived of support; he also records the fact that the plane of the epiphysial line tends to become more vertical at the start of the second decade.

Willis[3] (1929) leaves etiology with the statement that "the majority of his cases have occurred following definite injury in overweight adolescents of the infantile type." This includes all three accepted factors and implies that they may all operate in the same case.

Irwin[5] (1929) mentions thinning of the periosteum and alteration of the plane of the epiphysial line towards the vertical, but does not agree with either of them. He states that up to ten years of age the cartilages of head and greater trochanter are connected and that it is wellnigh impossible to separate one, without the other, by force. This seems to imply a belief in trauma as the main etiological factor. *Case* 8 of this series is interesting from this point of view, because in it the head of the femur in a child aged 4 was forced out of the acetabulum and the greater trochanter left behind in the normal situation (*see Fig.* 232).

Jahss[6] (1931) makes no statement on the question of etiology. Perkins[7] (1932) points out that the flake of diaphysis, usually separated with other epiphyses, is not seen in the case of the head of the femur. This is true for all the X-rays studied, but, in the writer's opinion, cannot be used as evidence to rule out trauma from the etiology: it merely indicates that the degree of force producing the lesion in the hip is much less than that which displaces other epiphyses. It is definite evidence, however, that there is normally a secure attachment between epiphysial cartilage and metaphysis, and suggests that there is some weakening of this attachment, in the case of the head of the femur, before the displacement commences.

With regard to my cases, in four of them I was able to verify the history of trauma from the patient's parents or from other responsible people, and in each one of these there was a definite history of an *indirect* injury, i.e., the force had been applied to the knee or foot. It is interesting to contrast these with *Cases* 8 and 9, in which there was an equally definite history of direct injury to the region of the hip-joint and in neither of which did a typical slipped epiphysis occur. I feel that this fact is of importance and is of help in advancing an hypothesis of the etiology.

Two cases had generalized epiphysial involvement, and the X-ray findings were consistent with what has been termed 'renal rickets', and the patients were in the right age group.

It seems reasonable to suppose that a young patient would easily forget a mild trauma to the knee when questioned regarding the hip. From a consideration of these facts I have come to the conclusion that, with

regard to the etiology, cases of slipped epiphysis can be divided into two types :—

1. Those with definite evidence of disordered internal secretion in which there will be other epiphyses involved besides that of the head of the femur.

2. Those with a history of indirect trauma, however small, where only the capital epiphysis of the femur is implicated and where there are no other signs of disease.

Once the epiphysis has 'gone over the brink', it seems reasonable to conclude that body weight will have a very definite effect in increasing the deformity, and this is probably the explanation of Key's statement that the three factors are never found together.

The 'overweight' type of case, so frequently referred to in the literature, but not so often seen, should be placed in the second class, with emphasis on the fact that the increased body weight is a secondary factor. Most of the recorded cases are unilateral.

It remains to consider whether there is any connecting link between these two types, and if so, what the nature of it is.

The kind of injury, and the interval between it and the onset of the symptoms and signs, which is noteworthy in the cases cited, suggests the tearing of periosteum and blood-vessels rather than damage to bone or cartilage. The natural consequence of such damage would be a reactive hyperæmia. Further, the acetabulum grips the head of the femur so firmly that any strain applied must fall on or near the region of the epiphysial line.

In bone, hyperæmia produces decalcification (Leriche and Polichard[8]), and I think evidence of rarefaction is to be seen in the early X-rays of these cases, at a time when it cannot be accounted for by immobilization, in the metaphysis. In rickets and in 'renal rickets' it is known that decalcification occurs in the metaphysial and epiphysial regions.

We are therefore brought back to the statement of Kocher[9] in 1894, quoted by Key, Irwin, and de Quervain,[11] that the underlying factor in slipping of the femoral epiphysis is a 'localized osteomalacia' of the neck of the femur. The decalcification occurs in the area of most recently formed bone and thus weakens the attachment of epiphysial cartilage to metaphysis.

As mentioned above, there is no flake of diaphysis detached with the epiphysis because the original force is small and does not produce any displacement of itself.

The explanation of the cases quoted as having no history of injury is that the injury was slight and not considered worthy of notice by the patient. It is interesting to note that in the older literature up to 1907 this condition is always referred to under the heading of 'traumatic coxa vara', although the authors Kocher,[9] Kirmisson,[12] and Elmslie[4] all emphasize the small amount of force recorded in the case histories. It seems that later writers, impressed by the minimal trauma, decided for that reason alone that the injury was of no significance in the etiology.

Either trauma or endocrine disturbance, therefore, produces a weakening at the metaphysis which allows the epiphysis to slip gradually under the influence of either body weight or muscular action.

The three factors are, therefore, accounted for, and the reason why they are never all present together is apparent—body weight cannot act until the process has been started by one of the other factors.

TREATMENT OF SLIPPED EPIPHYSIS.

The first stage consists in placing the patient on a well-made and accurately fitted frame (*Fig.* 234) which is constructed in the manner of a Jones spinal frame with extension ends to the leg pieces. Studs are provided on the pelvic bars for the attachment of a groin strap. It will be noticed that no provision is made for abduction of the lower limbs.

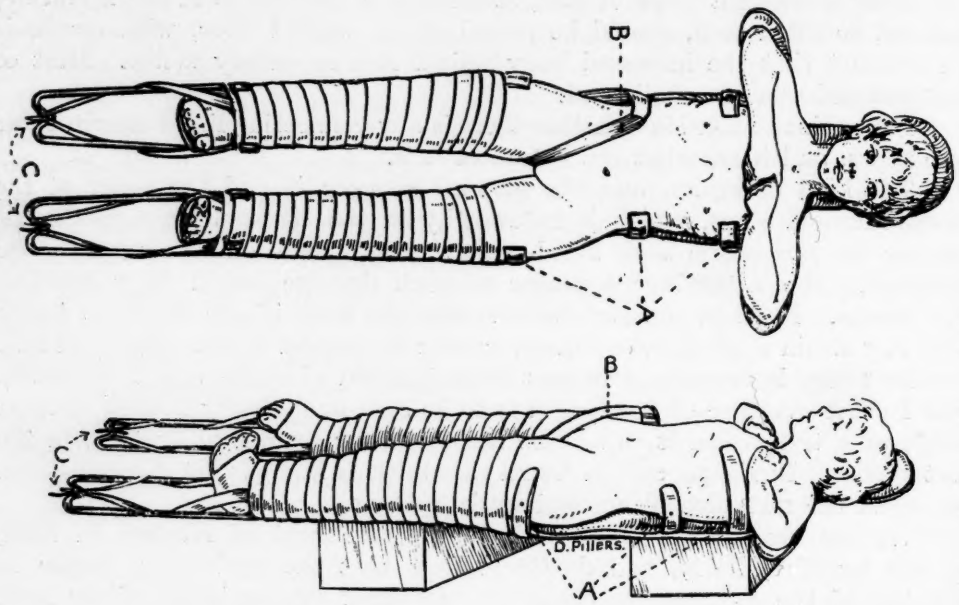

FIG. 234.—Illustrating position of patient on frame. A, Side bar of frame; B, Groin strap *on side of lesion*; C, Extension ends, with tapes tied. Note blocks, which facilitate nursing.

Strapping extensions, with tapes attached, are then applied to both legs from the groins to the malleoli, as used for fixed traction in a Thomas bed knee splint. A groin strap is now fixed *on the side of the lesion*, by means of two studs riveted to the side wing of the frame. Traction is then applied by tying both sets of extension tapes over the extension ends of the leg-pieces, that on the sound side being used to steady the pelvis. Finally both limbs are bandaged to the frame, and on the affected side this bandage is used gradually to correct the external rotation which is generally found to be present.

It will be seen that the result is steady and increasing traction applied to the affected hip in a relatively adducted position, i.e., the shaft and neck of the femur are being pulled in an axis parallel to the vertical axis of the body, while the groin strap, and traction on the unaffected leg, provide two counter-balancing forces which prevent both adduction and abduction at the injured hip-joint.

A most important part of the treatment is the nursing, and it is a steady application of simple routine principles which is necessary. All pressure points must be rubbed with spirit until dry, and then powdered, once every four hours day and night, and extension tapes tightened at the same time. Particular attention must be paid to the groin from which counter-pressure is being taken. Control X-rays are taken at intervals of fourteen days until the epiphysis has slid back into place, and traction is continued for an arbitrary period of one month after this has happened.

For the second stage the patient is removed from the frame but is kept recumbent and is allowed free *active* non-weight-bearing exercises at the hip-joint. This is continued until the range of movement no longer increases, and, as will be seen from the case reports, no definite period can be allotted. But as the patients are young, and time is of relative unimportance, it is much better to prolong the period and make certain that the reposition has become stable.

In the last stage of treatment the patient is allowed up in a walking caliper splint, and this must be worn for at least one year from the time that the treatment was commenced. Exercises in recumbency may be continued with advantage.

Case 1.—J. R., male, aged 15 years. First seen on July 2, 1931, and gave the history that a month earlier he had fallen off a bicycle and struck his left knee on the ground. One week after the accident he commenced to have pain in the left hip and became unable to stand on the affected leg. It was noted that his general condition was good but that he was definitely overweight and overheight for his age.

July 2, 1931.—X-ray shows condition of left hip (*Fig.* 235). He was placed on a frame in the manner described.

July 23.—X-ray shows partial reduction (*Fig.* 236).

Aug. 11.—X-ray shows that reduction has proceeded, and the margin of the head is now continuous with the upper margin of the neck (*Fig.* 237).

Sept. 25.—X-ray shows reduction is now complete, the criteria being: (1) Shenton's line is normal; (2) The epiphysis has assumed its normal elliptical shape instead of the sphere of the earlier films. This film also shows the development of new bone at the lower end of the epiphysial line where the periosteum was stripped off by the original trauma (*Fig.* 238). The patient was now taken off the frame and allowed free active movements in recumbency.

Oct. 3.—The condition noted was: No pain; about 20° active movement present in all directions; Bryant's triangle showed a small amount of real shortening, less than ½ in.; considerable wasting of gluteal muscles.

Oct. 10.—Confirmed reduction (*Fig.* 239).

Oct. 24.—Shortening was still just measurable. Movement had increased to: Abduction, 45°; flexion, 45°; adduction, full; external rotation, full; internal rotation, nil. There was noticeable recovery in gluteal muscles.

Nov. 7.—It was curious that shortening had now disappeared, although traction had been stopped for some time. Abduction, 45°; flexion, 45°; adduction, full; external rotation, full; internal rotation, half range.

Feb. 20, 1932.—Flexion had increased to a right-angle, other movements remained the same. The patient was fitted with a walking caliper and exercises continued as an out-patient.

Sept. 10.—Abduction, full; flexion, 90°; adduction, full; external rotation, full; internal rotation, limited.

January, 1933.—This patient has been actively employed for the last six months and has now applied for admission to the Police Force.

Fig. 235.

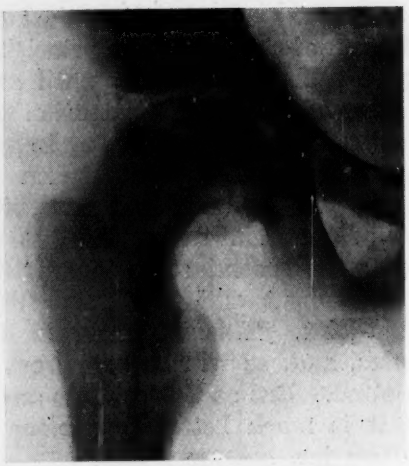

Fig. 236.

Fig. 237.

Fig. 238.

Fig. 239.

Figs. 235–239.—*Case* 1.
X-rays showing progress of treatment.
X shows areas of rarefaction.

Fig. 235.—July 2, 1931.
Fig. 236.—July 23, 1931.
Fig. 237.—Aug. 11, 1931.
Fig. 238.—Sept. 15, 1931.
Fig. 239.—Oct. 10, 1931.

Fig. 240.

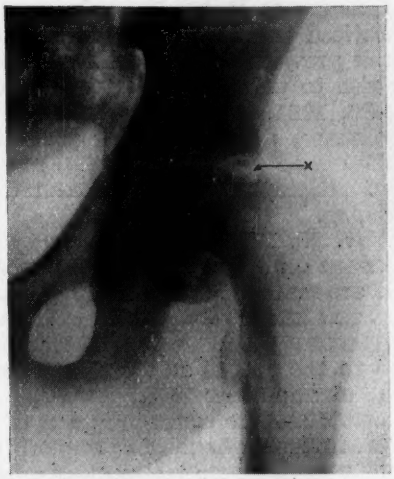

Fig. 241.

Fig. 242.

Fig. 243.

Fig. 244.

Fig. 245.

Figs. 240–245.—*Case* 3. X-rays showing progress of treatment.
X shows areas of rarefaction.

Fig. 240.—Nov. 20, 1931. Fig. 241.—Dec. 22, 1931. Fig. 242.—Jan. 23, 1932.
Fig. 243.—Feb. 27, 1932. Fig. 244.—April 2, 1932. Fig. 245.—July 19, 1932.

Case 3.—C. R. W. H., male, aged 14 years. One month previously the patient had noticed 'growing pains' in the left hip and had been noticed to limp a little. A week previously he had knocked his left ankle while going upstairs; and this gave rise to pain again; later he found he could not walk. When examined on Nov. 20, 1931, the leg was held everted and abducted at the hip-joint and there was a tense, painful swelling about the size of an egg in Scarpa's triangle. There was 20° active flexion at the hip but all other movements were limited by muscle spasm. Attempted movement caused pain. On measurement there was 1 in. real shortening.

Nov. 20, 1931.—X-ray shows typical slipped epiphysis (*Fig.* 240). The patient was placed on a frame in the manner described. Unlike *Case* 1, all the difficulties which are made so much of by the opponents of fixed traction were encountered, but as will be seen by the notes below they were all successfully overcome without resort to any other method of treatment; furthermore, the patient was never subjected to the risk of an anæsthetic, nor was the hip traumatized by violent manipulation at any time.

Dec. 10.—The commencement of a pressure sore was observed in the left groin and the inversion bandaging around the leg had produced œdema of the foot. The patient was therefore taken off the frame. In order that no ground might be lost, the foot of the bed was raised and the patient suspended by tying the extension tapes to the bed rail; a pillow was placed behind the knees.

Dec. 19.—Real shortening was still 1 in., and the pressure sore was not healed. A weight was attached to the extensions of the left leg to prevent any shortening while off the frame.

Dec. 23.—Control X-ray showed no change in the position (*Fig.* 241). As the sore had now healed, the patient was put back on the frame, but very light traction was used to start with and the strap kept on the right side temporarily.

Jan. 9, 1932.—A right foot-drop developed and the strapping was removed from the area of the external condyle of the femur and the head and neck of the fibula.

Jan. 16.—The groin strap had now been replaced on the side of the lesion and power had returned to the right anterior tibial group of muscles.

Jan. 23.—X-ray showed marked improvement in the position of the epiphysis (*Fig.* 242).

Feb. 20.—X-ray showed the epiphysis almost in its normal position. Measured by Bryant's triangle no shortening was recorded (*Fig.* 243).

March 19.—Removed from frame. Active movements as follows were noted immediately the patient was free: Abduction, 30°; flexion, 25°; little rotation. Active exercises in recumbency were commenced.

April 2.—X-ray confirmed position (*Fig.* 244).

May.—During May the patient was allowed to walk in a caliper.

July 16.—X-ray showed the position to be maintained (*Fig.* 245). Active movements: Abduction, 30°; flexion, 90°; external rotation, full; internal rotation, half range.

January, 1933.—The patient is now walking well without his splint.

Case 11.—A. G. H., male, aged 17 years. This youth was first seen on April 13, 1933. He stated that he had noticed that he was walking with a limp for eight weeks previously, and that his left knee and ankle 'gave way' easily. There was no history of any previous accident or illness.

On examination he was found to lie with the left leg externally rotated at the hip, and there was ¾ in. real shortening present. Abduction and internal rotation were limited at the hip-joint and there was 1 in. wasting of the quadriceps 4 in. above the superior margin of the patella. X-ray (*Fig.* 246) shows slipping of the left upper femoral epiphysis. (Compare with X-ray of normal hip, *Fig.* 247.)

Fixed traction, as described earlier, was commenced in a few days, as soon as a frame could be obtained, and a series of X-rays taken during treatment. *Fig.* 248 shows the condition after three weeks' traction. The relationship between capital epiphysis and femoral neck has definitely altered. The outline of the head and

upper margin of the neck are no longer continuous and Shenton's line is nearer to the normal. *Fig.* 249 was taken after six weeks' traction. Correction has progressed and Shenton's line is now normal. Skiagrams taken at the end of eight

FIG. 246.—*Case* 11. Condition when diagnosis was made.

FIG. 247.—*Case* 11. Skiagram of normal hip.

FIG. 248.—*Case* 11. Condition after traction for three weeks.

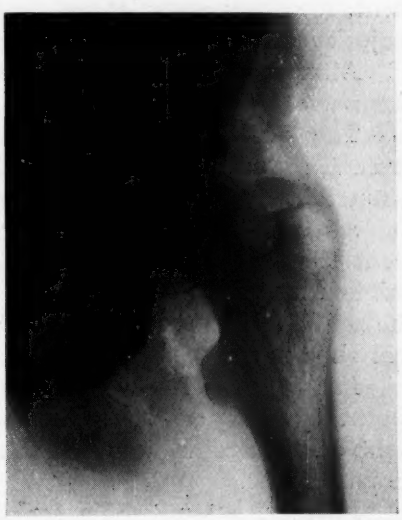

FIG. 249.—*Case* 11. Final condition on removal of frame.

weeks showed no further correction, and clinical measurement confirmed full length of the limb. The patient was thereupon removed from the frame.

Active non-weight-bearing exercises were then started, in recumbency, and have been continued to the present time. The range of movement at the left hip

is now: Flexion, 90°; abduction, 45°; rotation, the foot can be rotated internally as far as the foot of the normal leg. Measurement shows that full-length is maintained, and there is no pain. The patient has now been measured for a walking caliper.

SURVEY OF METHODS AND RESULTS.

Key[1] records eight cases in which either manipulation or operation were used; 50 per cent of these had shortening of 1 in. or more afterwards. Half of them had a flexion deformity and 45° was the maximum of abduction obtained. He rightly concluded that forcible reduction of the displaced epiphysis by the Whitman method was a fertile source of stiff hips subsequently, and recommended reduction by an open operation in cases where bony consolidation had not occurred.

Badgely[2] followed up Key's suggestion, but out of six cases reported as operated on, two were of the Whitman reconstruction type, one was an osteotomy, and two of the remaining three developed non-union. Also the standard of comparison was not high, as one case with ¾ in. shortening was quoted as a good result.

Willis[3] later in the same year recommended manipulation, but gives no definite after-results. Irwin[5] does not record any after-results. Jahss[6] records three cases in detail and describes his own modification of the method of reduction by forcible manipulation. His results are good and he shows X-rays confirming the reduction in each case. Taylor[10] quotes a series of Liverpool cases which he had collected. Fourteen of these were old enough to be considered after-results, and included cases which had had no treatment, forcible manipulations, and reduction by open operation. The great majority of the results were poor.

After a consideration of these records the following points suggested themselves:—

The methods in use for the reduction of this deformity, up to the present, are open to serious criticism on the grounds that they do not produce a functionally useful hip, and function is the only real test of success.

Some definite standard of result must be laid down at which the treatment should aim, and I would state it thus: The patient should have a painless hip and a minimum of 90° active flexion and 45° abduction before the result is considered good. Further, there should not be more than ½ in. real shortening.

Far too much emphasis has been laid upon anatomical reduction as shown by X-rays, and the functional recovery of the hip-joint has been lost sight of. Anatomical reduction is worthless if the price of a stiff hip has to be paid for it.

Further, a study of a series of X-rays has convinced me that forcible abduction of the hip in these cases is wrong in principle and practice; as can be seen from *Fig.* 250, when the femur is abducted the lower edge of the metaphysis engages the separated epiphysial surface and tends to force the epiphysis even farther downwards and backwards in the acetabulum. In effect, this means that the leverage which is being used to effect reduction has its fulcrum at the point where articular cartilage of the head impinges

on the lower and posterior acetabular margin; the result is bruising of the cartilage and a subsequent arthritis of the traumatic type; and even when reduction starts, the metaphysial edge ploughs through the cervical surface of the epiphysis. Together with the tearing of capsule and ligamentum teres, mentioned by other authors, the damage is quite sufficient to account for subsequent stiffness.

Considered from the anatomical point of view alone, the close fit of the head of the femur in the acetabulum makes it imperative that the reducing force should be in the long axis of the femur and not in a direction which forces the head against the acetabular wall.

The paradox that "untreated cases of slipped epiphysis do better than treated cases", stated by Key[1], is capable of a simple explanation on these lines; in striving after perfect anatomical reduction, function has been quite forgotten. A similar conclusion is reached by Perkins[7] in his article.

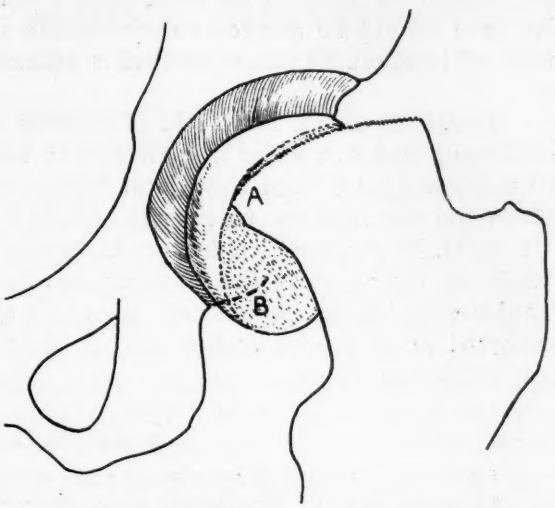

FIG. 250.—Line drawing from skiagram of recent case of slipped epiphysis. A shows point where lower edge of metaphysis engages separated epiphysial surface; B indicates fulcrum of leverage force when femur is abducted.

The idea of traction applied gradually, without abduction of the hip, has been evolved in response to the demands laid down earlier, and eliminates any possibility of damage to articular cartilage or capsule and blood-vessels, thus leaving the way clear for the future development of a movable and useful joint. At the same time the method holds out the prospect of at least as good a reduction as claimed for other methods, although this is not made the criterion.

I do not agree with Perkins[7] that gradual traction should be confined to any particular type of case. It is worth a trial in all cases and a period of seven days is not long enough in which to judge of its efficacy. It is agreed that for the old case, consolidated in deformity, subtrochanteric osteotomy is excellent treatment, and *Case* 10 of my series bears this out, but it is surely wrong to fall back deliberately on to a second line of defence when simple traction is likely to succeed if persisted with.

Open operation is condemned by the statistics of those who have tried it.

SUMMARY.

1. The etiology of slipped epiphysis is considered, and it is suggested that either indirect trauma or endocrine disorder produces weakening at the epiphysial line; displacement then proceeds under the force of body weight.

2. Treatment by gradual traction in adduction is described and three successful cases are reported in detail with serial X-rays.

3. Gradual traction is advocated as being the most efficient form of treatment. Subtrochanteric osteotomy is an excellent second line of defence, but should be reserved for cases seen when the deformity has consolidated. No case should be allowed to consolidate in deformity without a prolonged trial of traction. Open operation is condemned.

I wish to thank Mr. T. P. McMurray for his guidance in the treatment of *Cases* 1 and 3 at Alder Hey Hospital; Mr. T. R. W. Armour for permission to publish *Case* 8 from the Royal Southern Hospital, Liverpool; Mr. W. J. Eastwood for facilities to examine *Cases* 2 and 4 at the Wigan Inflrmary; Mr. C. O. Davies for *Case* 5 from Liverpool Stanley Hospital; and Mr. C. A. Wells for *Case* 7 from the Clatterbridge Institution, Cheshire. Mr. V. J. M. Taylor very kindly allowed me access to a series of earlier cases which he had collected in Liverpool, and which he has published recently.

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OBSERVATIONS UPON MULTIPLE INTRAMESENTERIC DIVERTICULA OF THE SMALL INTESTINE.

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The condition of multiple intramesenteric diverticula of the small intestine is a definite pathological state, which must be distinguished from others in which diverticula occur in the small bowel at its mesenteric border, such as traction diverticula secondary to adhesions or to the scarring of old inflammatory lesions, congenital pouches associated with accessory gland tissue, and vestigial and developmental cysts and diverticula.

Published records, both in this country and abroad, are rather scarce, and many are insufficiently thorough in details of the patient's past history and symptoms and in the cause of death to be of any assistance in studying the subject as a whole, consisting only of a bare description of the naked-eye pathology.

This paper is based upon a detailed study of seven new cases collected by the writer. Three of these were observed personally as patients in the wards of St. Thomas's Hospital, and in two of these and in the four others material was available for pathological investigation.

For the purpose of a more comprehensive survey these seven have been considered together with thirteen others previously recorded with sufficient detail, both clinical and pathological, to assist in an investigation of the origin and significance of the condition. Of these thirteen, twelve have been described in various medical journals. These are listed under the names of the authors in the reference table at the end of the paper.[1–5, 8, 10, 11, 13–15] The thirteenth case is that from which was obtained specimen No. 3065 K.I. in the Museum of the Medical College, University College Hospital, where the case notes are also preserved. These thirteen, with the writer's seven, make a total of twenty fully recorded cases which have been used as a source of general and statistical information in compiling this paper.

In the present number of this Journal another writer (Ian Fraser: "The Diverticula of the Jejuno-ileum") has made a general survey of the whole subject (p. 183). It is therefore unnecessary to repeat a great deal that is already written, and in this paper the more general features of the condition will be described briefly first, and the writer will confine himself to a full description only of his own cases and of the conclusions reached by clinical and pathological study.

GENERAL CONSIDERATIONS.

The condition is rare. Often it has been found accidentally at the post-mortem examination of persons who have died from some totally different cause. Nineteen out of twenty mentioned by Terry and Mugler[15] were so

found. In such there is frequently no history of symptoms attributable to the condition of the bowel, and we are therefore led to wonder whether, as J. T. Case[5] suggests, the condition (though rare) may not be somewhat more common in elderly persons than is usually supposed.

In the twenty cases upon which this paper is based the average age at which the condition was discovered is 64, and the writer can find no description of multiple diverticula of this type being found in a child or young person. In some of these cases the diverticula were no doubt of long standing, but in most of the younger cases the diverticula were few in number, and it is evident that the condition is developed typically in persons past middle age, and probably seldom becomes apparent before the age of 40 at least.

The twenty cases in this series comprise fourteen men and six women.

THE GROSS ANATOMY OF THE CONDITION.

Once seen, this condition can never be mistaken, so remarkable is the appearance when well developed (*Figs.* 251, 252). A number of diverticula are seen in the jejunum and upper part of the ileum. They are always most plentiful in the first two or three feet beyond the duodenojejunal junction, and when only two or three are present they are found in the first jejunal loop. When a very large number occur—as many as four hundred have been recorded—they sometimes extend for several feet along the bowel, and at times are scattered throughout almost the whole length of the ileum as well as the jejunum. In such, however, the larger diverticula lie close to the duodenojejunal junction.

In the same specimen the diverticula may vary in size from small conical pockets the size of a pea to large globular diverticula 7 cm. or more in diameter (*see Figs.* 251, 252). All except the very small sacs have obviously thin walls, though here and there the larger sacs may be thickened by fine adhesions to the adjacent bowel.

The diverticula all occur at or near the mesenteric border. The small ones push straight into the mesentery, but as they increase in size they bulge out from the side of the mesentery, usually towards the right. Finally, the very large diverticula tend to stand away from the mesentery and to overlap the bowel, completely covered by a peritoneal coat except at their broad base of attachment.

Klebs[12] in 1867 was the first writer to point out that there is a definite relationship between the mesenteric blood-vessels and the diverticula. Each diverticulum arises from a point at which one of the vasa recta of the superior mesenteric artery reaches the bowel wall.

In the case of the small diverticula this is readily made out (*see Fig.* 252), but with the very large diverticula this relationship to the vessels is lost, as the latter are pushed to one side by the enlarging sacs. The exact significance of the vessels in the mode of origin of the diverticula will be discussed later. An interesting feature is that there are usually more diverticula to the right of the mesentery than to the left. Probably this is because there is more room for them towards the right, since the first loop of the jejunum lies over towards the left and its mesentery is in contact on the left with the rigid

structures forming the posterior wall of the peritoneal cavity. Where the diverticula appear for more than a couple of feet down the jejunum this partiality of the diverticula for the right side of the mesentery disappears, which fits in well with this explanation.

The orifices of communication between the bowel and the diverticula are large. The smaller diverticula, 1 cm. across, have orifices opening into the bowel almost as wide as their own greatest diameter. A larger diverticulum, 2·5 cm. in diameter, will have an orifice perhaps 1·5 cm. wide. The largest diverticula, 6 or 7 cm. in diameter, have orifices 2 to 3 cm. wide.

FIG. 251.—Sketch of part of specimen from a case described by Mr. L. R. Braithwaite. Shows the great size to which diverticula of this type can grow, these being easily the largest that the writer has seen in any specimen of this condition. Small commencing diverticula are also seen. (× ¾.) *(Specimen in Museum of the Royal College of Surgeons.)*

THE CLINICAL STATE.

Clinically, patients fall into three groups :—

1. Those in whom the condition is discovered purely by accident—perhaps post mortem—and in whom the state of the bowel has produced no symptoms during life and is in no way directly related to the cause of death.

2. Those in whom there are definite symptoms produced by the diverticula during life.

3. Those in whom symptoms, if they have been present at all, have been comparatively slight, but who are seen because of an urgent abdominal emergency directly related to the presence of the diverticula.

Twelve of these twenty cases fall into Class 1 in that there is no history of any symptoms definitely attributable to the condition previous to its discovery. The first of the writer's new cases is an example of this type.

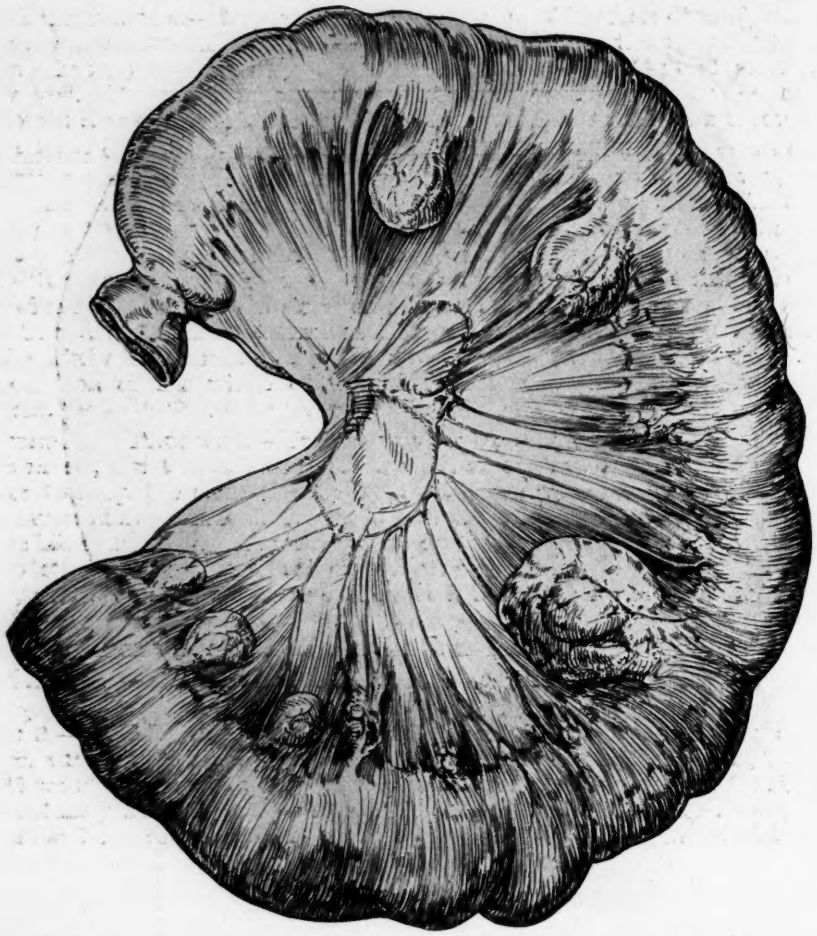

FIG. 252.—*Case* 1. The first 16 in. of the jejunum seen from the right. Diverticula of many different sizes are seen, and the direct relationship of the vasa recta to the points at which they arise is in some cases clearly evident.

Case 1.—A. S., female, aged 65. Admitted to Lewisham Hospital in January, 1926, suffering from a vaginal recurrence of carcinoma of the body of the uterus, for which a hysterectomy had been previously performed elsewhere. A bleeding mass was present in the vaginal scar. Within a few days this patient developed subacute intestinal obstruction of large-bowel type. General peritonitis rapidly supervened and the patient died.

AUTOPSY.—At the post-mortem examination the cause of the obstruction was

found to be a carcinoma of the sigmoid colon. A stercoral ulcer of the cæcum had perforated. There were a number of diverticula in the sigmoid colon.

In the upper 2 ft. of the jejunum were some twenty diverticula at the mesenteric border, varying in size from a small pea to a large walnut. *Fig.* 252 shows the specimen of the jejunum from this case. The relationship of the vessels to the diverticula is well shown. The histology of this specimen will be described later.

FIG. 253.—*Case* 2. X-ray six hours after bismuth meal, showing multiple fluid levels in the diverticula, many with gas above. The diverticula in this case were many of them large and they were distributed throughout the whole of the jejunum. Probably hundreds were present.

During life these diverticula were quite unsuspected, and any dyspeptic symptoms that may have been present were not sufficiently severe to attract attention.

(For the clinical details the writer is indebted to Dr. H. Nockolds, Medical Superintendent of Lewisham Hospital.)

On the other hand, eight cases in the series of twenty show certain chronic symptoms attributable to the abnormal condition of the bowel during life. The writer's second case is of this clinical type.

Case 2.—H. L., male, aged 63. Admitted to St. Thomas's Hospital under the care of Dr. Cassidy. Two years' history of abdominal discomfort and wasting. This began as slight constant flatulence; always feeling as if he wanted to "bring up wind", with a certain amount of mild aching pain in the abdomen. As time went on the flatulence became worse, and he began to vomit, at first only occasionally, but much more frequently for the last six months. The more frequent vomiting was often preceded by acute attacks of pain across the upper part of the abdomen. Of late the vomit had contained a good deal of undigested food, and had occurred at all hours of the day and night. Vomiting did not now relieve the pain completely. For the last six months particularly he had been losing weight rapidly. For several months he had also been very constipated.

On Examination.—Very wasted; edentulous. Abdomen diffusely tender and showing an indefinite fullness in the upper part towards the right. This fullness was somewhat hyper-resonant, suggesting dilated bowel. General arteriosclerosis very marked. Pulse 80, blood-pressure 120/90. An X-ray following a bismuth meal showed a very remarkable appearance of multiple fluid levels throughout the small intestine (*Fig.* 253).

Operation.—A laparotomy was performed, revealing the following state of affairs: The small bowel generally was slightly dilated, especially in the upper part. A great many, probably hundreds, of diverticula were present in the small bowel at the mesenteric border. They varied in size from a pea to a tangerine orange. The larger diverticula were in the upper part of the small bowel, and they gradually lessened in their average size as the bowel was examined downwards. The diverticula contained mainly gas. No short-circuiting operation that would be likely to benefit the patient was possible, and he was considered unfit for an excision of the whole of the affected bowel. The abdomen was therefore closed.

Subsequent Progress.—Healing was rapid, and with rest in bed, small and frequent meals, strict attention to the bowels, and the taking of small doses of carminatives to keep the stomach free from distension with gas, the patient gradually became much more comfortable. Alkalis in the form of bismuth carbonate helped to relieve his symptoms. About a month after the operation, however, the patient began to show signs of cerebral degeneration of a senile type. This man was, however, still alive in comparative comfort six months later.

Another example of rather similar but less marked symptoms is shown in the following—the third of the writer's cases.

Case 3.—W. W., male, aged 61. Had complained for "some months" of flatulence, vague abdominal pain, and a feeling of fullness after meals, but no real pain. There had been no vomiting. He had been rather constipated for six months. He had actually been increasing in weight and getting stouter for several months. Two months before his death he went to his doctor complaining of these vague symptoms, but the only discoverable signs were very marked general arteriosclerosis and a pronounced degree of myocardial degeneration. One day, while leaving a train at Waterloo Station, he suddenly fell dead.

Autopsy.—At the post-mortem examination (held at St. Thomas's Hospital) there was found marked general arteriosclerosis, with atheroma of the aorta and much narrowing of the coronary vessels. The first 4 ft. of the jejunum showed about fifty diverticula.

J. T. Case[5] in one of his patients found a history, very similar to the above, of mild 'indigestion' for many months; a feeling of pressure and distension following meals, relieved somewhat by belching. No nausea or vomiting. No real pain, but rather abdominal discomfort. For six months there had been much intestinal flatulence.

Diagnosis.

The abdominal symptoms are not distinctive, and in the absence of any X-ray evidence of the diverticula it is certain that a diagnosis of some much more common condition would be made. In *Case* 2 the symptoms suggested simply a gradually increasing degree of small-bowel obstruction. In such cases X-rays following an opaque meal would seem to be the only way of making a certain diagnosis other than by laparotomy.

X-ray Diagnosis.—J. T. Case claims to have diagnosed five cases in this way—two of them checked by laparotomy. Gas accumulation in the diverticula was seen on the screen before giving the bismuth meal in some of his cases.

Fig. 253 shows very strikingly indeed the multiple fluid levels with gas above them six hours after a bismuth meal, and shows the condition in *Case* 2 described above. The diverticula were seen to be almost clear of bismuth ten hours after the meal, and the small bowel appeared to empty within the normal time limit. This bears out Case's statement that the diverticula take eight to ten hours to empty, and checks the supposition which we have already made on anatomical grounds that the wide openings of the sacs would not retain the fluid bowel contents for long. Peristaltic movements of the bowel pressing on the thin-walled diverticula would doubtless empty them easily of their fluid contents, and it is perhaps an accumulation of gas, rather than of fluid bowel contents, which gives rise to the feeling of fullness of which the patient complains.

Having once seen the typical X-ray appearance of fluid levels in the diverticula with gas accumulation above as in *Fig.* 253, it would be imagined that the diagnosis would easily be made by X-rays whenever the diverticula were large and numerous. Curiously enough this is not always so. In *Case* 7, to be described later, repeated X-rays taken after recovery from the operation for peritonitis failed to show clear evidence of any diverticula, though more than a dozen large sacs were known still to be present. Other writers have noted this in individual patients, and it is probable that the amount of retention in the diverticula is much greater at some times than at others, varying perhaps with the tone of the involuntary muscle of the bowel wall.

Treatment of the Uncomplicated Condition.

If found accidentally by X-rays in a person in whom it is producing no symptoms, the condition should be left alone without surgical interference. If troublesome symptoms are present, an exploratory operation is certainly justifiable, as in some cases, such as that described by L. R. Braithwaite,[3] a complete resection of the affected area of bowel may be possible, and this is undoubtedly the most satisfactory treatment where it is practicable, for, as we shall see, acute complications of the condition may arise without warning. Should the diverticula be too widely scattered for this, a short-circuit operation consisting of an anastomosis of the upper and lower ends of the affected area may relieve the symptoms to some extent and serve as a prelude to a later excision when the patient shall be in a better state to stand the more severe operation.

Inversion or excision of individual diverticula, as Gordinier and Sampson[8] point out, is likely to endanger the blood-supply of the bowel wall locally, and is obviously impracticable as a radical treatment where there are a great number of diverticula.

The treatment if acute complications should arise will be discussed later.

If symptoms have only recently begun to appear, it is worth while trying a regulation of the diet, so that meals are small in bulk.

The bowels should be regulated and kept freely open. In the cases which the writer himself has seen, alkalis in the form of sodium bicarbonate and bismuth oxycarbonate in a simple mixture give relief to the discomfort. Carminatives of any sort seem to help; and, theoretically, tonics and stimulants to the muscular action of the bowel wall should be of value. Rest in bed alone appears to help some patients, and indeed simply lying down for an hour often relieves completely a severe attack of discomfort.

ACUTE COMPLICATIONS.

The gross anatomical abnormality present in many of these cases might lead to the expectation not only of the production of chronic symptoms, but also the sudden advent of acute complications. We have seen, however, that in many there are no chronic symptoms at all, and in the same way sudden disasters due to the presence of these diverticula appear to be rare.

The large size of the openings of the diverticula into the bowel is probably the significant feature in this freedom from complications, for there is very little retention of the bowel contents, which are fluid, and, incidentally, comparatively sterile, in this part of the intestine, so that no inflammation is set up in the walls of the diverticula. When an inflammation does arise, it seems to start in the loose connective tissue lying in the mesentery around the neck of a diverticulum. There is often a considerable bulk of loose fatty tissue lying here, especially immediately above and below the neck of the sac where the peritoneum bridges across the corner between the bowel and the diverticulum.

Here, too, the peritoneum is often thickened by many fine adhesions across from the sac to the bowel. These intramesenteric spaces, often of considerable size and filled with loose areolar tissue and fat, are separated from the lumina of the larger diverticula only by thinned mucous membrane. In Gordinier and Sampson's case this region had been the site of an inflammation which had obliterated the neck of a diverticulum. In three of the four new cases about to be described infection with perforation into the peritoneum had occurred at this point. In the fourth the site of the commencing inflammation could not be identified.

The writer's four cases of acute inflammation arising in this condition are as follows:—

Case 4.—Female, aged 72 (February, 1923).

HISTORY.—Occasionally constipated, but bowels usually regular. Awoke one morning at 5 a.m. with sudden acute pain in abdomen, localized to the left side just above the umbilicus. Vomited once; abdomen rigid, slightly distended. Twelve hours later pain was less severe and vomiting had diminished.

On Examination.—Pulse 120, temperature subnormal. Tongue dry and brown. Abdomen motionless and rigid. Acute tenderness to left of umbilicus. No loss of liver dullness. Diagnosis: perforation of some hollow viscus.

Operation.—Laparotomy. Small quantity of turbid fluid escaped on opening the abdomen. On left side, deep to tender area, was a perforated diverticulum in the mesenteric attachment of the upper part of the small intestine. In the adjacent part of the bowel on either side were several more diverticula, all at the mesenteric attachment. Two feet of the intestine were resected, and continuity restored by axial anastomosis. Adjacent portions of the intestine showed further diverticula. Recovery was complete and uneventful.

The perforation had occurred at the junction of the sac and the bowel wall, where a suppurative inflammation extended from the peritoneum as far as the mucous membrane, but the mucous membrane itself around the perforation showed but slight signs of acute inflammation. The wall of the sac consisted of fibrous tissue, containing only traces of muscle between the peritoneal and mucous coats. In the bowel wall the muscle was normally developed. (Described by permission of Mr. C. R. Nitch.)

Case 5.—Male, aged 56 (Aug. 24, 1928).

History.—Was accustomed for many years to suffer from "bilious attacks" with abdominal pain, vomiting, and diarrhœa, recurrent almost weekly, but these had ceased for twelve years. No indigestion since. Twelve hours before admission to hospital sudden epigastric pain stopped him working; he belched up wind at once. Three hours later he began to vomit. Pain was worse on taking a deep breath.

On Examination.—Pulse 70, temperature 98°, respiration 20. Tongue furred and dry. Tender and rigid between costal margin and umbilicus, whole belly rigid. Liver dullness much diminished.

Operation.—Laparotomy. Gas and blood-stained fluid in peritoneum. Stomach and duodenum normal. About 1 ft. of the upper jejunum found to be hyperæmic, with hæmorrhages into the peritoneum and mesentery. Five large diverticula at mesenteric border; one of these had perforated. All this portion of gut was resected, and end-to-end anastomosis was carried out.

In the course of the next three days paralytic ileus developed and failed to respond to all palliative treatment. A jejunostomy was performed under local anæsthesia, the tube being passed into the bowel below the line of anastomosis and up the lumen through the anastomosis. The tube drained well, but the patient died on Aug. 28. (Clinical details supplied by Mr. D. C. Taylor, Lewisham Hospital, and specimen loaned by Dr. Barnard, late Curator of Medical College Museum, University College.)

Case 6.—Female, aged 72 (March 4, 1928).

History.—Always very healthy. No history of any sort of abdominal trouble. A wiry old lady. Thirty-six hours before admission she suddenly had a severe attack of vomiting. Pain in left side of abdomen, high up at first, and later generalized all over. Could keep no food down. Bowels open twice.

On Examination.—Abdomen distended. There was general tenderness and rigidity. Pulse 100, temperature 98°. Nothing abnormal was felt per rectum.

Operation.—Laparotomy. Free turbid fluid in peritoneum. Appendix normal. In the left iliac fossa close to the sigmoid colon was a coil of small bowel bent round and adherent to itself. On straightening out this coil a small abscess was found in the concavity of the loop about a perforation in the neck of a diverticulum. This was one of seven diverticula in the upper 2 ft. of the jejunum, all running into the mesentery from the mesenteric border of the bowel. The mesentery was very œdematous and injected. About 9 in. of the jejunum were resected and end-to-end anastomosis obtained (*Fig.* 254).

For two days after operation the patient's condition was satisfactory, but she then began to sink gradually, becoming slowly more and more comatose.

Finally suppression of urine took place and she died with symptoms of uræmia on March 10.

Microscopy showed acute inflammation of the whole thickness of the wall of the diverticulum at the junction with the bowel.

AUTOPSY.—At the post-mortem examination five more diverticula were found in the jejunum, one being at the duodenojejunal junction, and the four others about 2 ft. below. (Surgical Unit, St. Thomas's Hospital.)

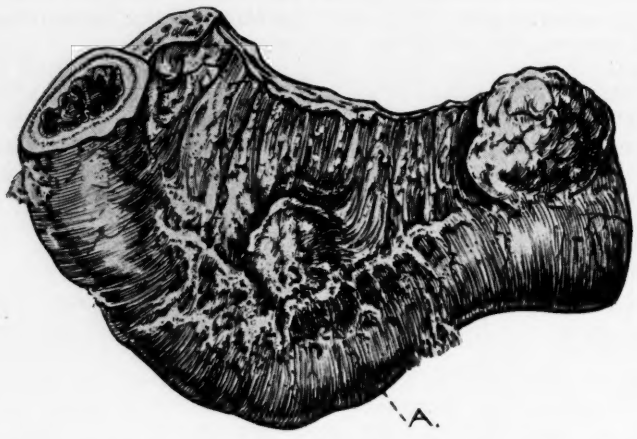

FIG. 254.—*Case* 6. Part of a loop of jejunum resected at operation for acute perforation of a diverticulum. The loop was found bent round and adherent to itself over the site of the perforation (A). Note the great œdema of the mesentery and the adjacent bowel wall, and the adherent fibrinous coagulum.

Case 7.—Male, aged 64 (March 5, 1928).

HISTORY.—Quite blameless; no abdominal trouble of any sort. A lean, wiry man. Sudden epigastric pain the day before admission, moving from side to side. Vomited several times.

ON EXAMINATION.—Abdomen distended and rigid. Pain continuous all over abdomen, worse in mid-line and umbilical region. Breathing laboured. Tenderness worse on left side. Nothing abnormal was felt per rectum.

OPERATION.—Laparotomy. Glairy pus free in the peritoneum. Jejunum inflamed, and of a bright pink colour for first 18 in., gradually fading off below. Both the bowel and the mesentery in this region were extremely œdematous. There were a great number of diverticula varying in size from a pea to a pigeon's egg, projecting into the mesentery throughout the inflamed area, and for several feet beyond. An indefinite mass was felt low down in the wall of the sigmoid colon, thought at the time to be an area of diverticulitis as it was not hard enough for a carcinoma. As there was no definite point of perforation visible, and as the patient's condition did not warrant any attempt at resection, the peritoneum was mopped out and the abdomen closed. Some suppuration occurred in the rectus sheath, but otherwise recovery was rapid and uneventful.

Subsequent examination with the sigmoidoscope and barium enemata failed to demonstrate any lesion in the sigmoid colon. A barium meal demonstrated the diverticula in the upper part of the small intestine. Since this operation the patient has suffered from a certain amount of flatulent dyspepsia, but has otherwise been in excellent health. (Surgical Unit, St. Thomas's Hospital.)

It is curious to note that as a remarkable coincidence these last two patients, both with the same rare complication of a rare condition, were admitted for urgent treatment to St. Thomas's Hospital on two consecutive days.

Discussion.—Unless the diverticula are already known to be present, diagnosis of an acute inflammation or perforation is clearly almost impossible. In these cases the preponderance of pain and rigidity on the left side is easily explained, but there are other more likely conditions that could produce this sign, and if the symptoms of the acute complication are the first produced by the condition, diagnosis will only be made at the operation. One of these cases manifested the symptoms of a subacute obstruction, another those of a perforated viscus, and the others those of a spreading peritonitis, but in none of them was there any doubt of the urgent necessity for operation.

As regards the actual procedure at operation, every case would have to be judged on its own merits. In four of the above cases resection was carried out; two recovered and two died. It is possible that the two last would have done better if the peritoneum had simply been mopped out and the area of the perforation buried by sewing over the surrounding tissue. If at the time of operation the condition of the patient is very critical, that would doubtless be the more hopeful procedure, for even under the best conditions a resection of a portion of the jejunum is not an operation that can be depended upon to give consistently happy results.

MINUTE ANATOMY.

The writer has had the opportunity of subjecting to microscopy tissue from four patients. In each case examination was made of sections of the bowel wall, of diverticula of various sizes, of the mesenteric vessels, and of any other available tissue that might be of interest.

The bowel wall between and opposite to the diverticula shows no apparent abnormality. Helvestine[11] has reported a reduction in the amount of the circular muscle of the bowel, even in parts of the wall not closely associated with the diverticula, but no evidence of this was found.

In almost every description of the microscopical examination of the walls of the diverticula it is stated that the muscle coats of the bowel are absent, so that the diverticulum consists only of mucous membrane and peritoneum separated by a little connective tissue and fat. The point is an important one. If this is true, it suggests that the diverticula are from the start herniations of the mucous membrane through apertures in the muscle, and any question of a traction effect from without by the vessels or by any other means is at once dismissed.

Evidently the descriptions are largely based on examinations only of the larger diverticula. In these admittedly muscle cells are few, though some evidence of the muscularis mucosæ persists in all but the very largest diverticula. On the other hand, if sections are made of small commencing diverticula, both longitudinal and circular muscle coats are found to be present and continuous, though admittedly somewhat thinned as if by stretching (*Figs.* 255, 256). This was the case in every commencing diverticulum examined by the writer.

In the larger diverticula—especially towards their apices—muscle cells are few and far between. Evidently when the diverticulum reaches a certain

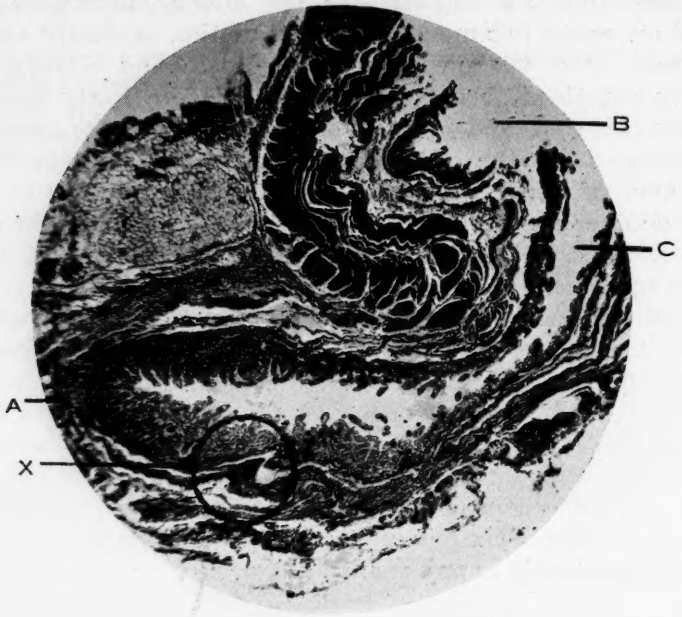

FIG. 255.—*Case* 1. Section of a small diverticulum 1·5 cm. in length cut in continuity with the adjacent bowel wall and showing the continuity of its lumen with that of the bowel. A, Apex of diverticulum; B, Lumen of bowel; C. Lumen of entrance of diverticulum; X, Area shown further enlarged in *Fig.* 256. The diverticulum is lying between the layers of the mesentery. (× 8.)

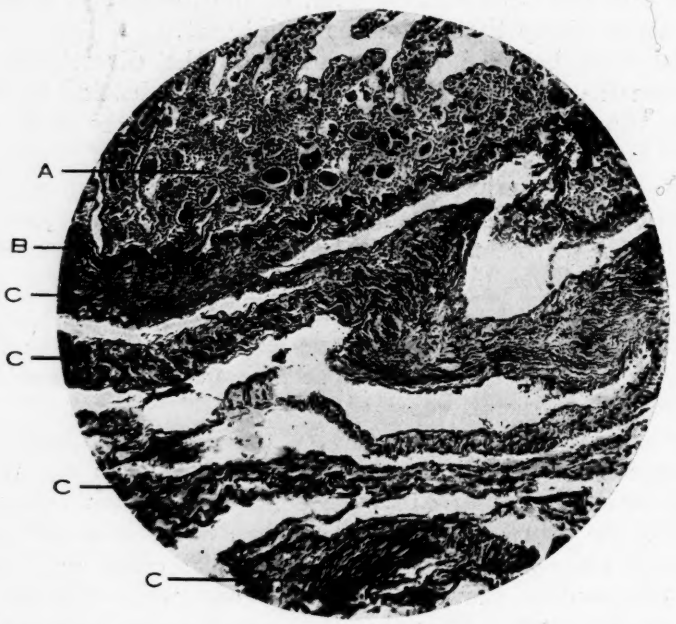

FIG. 256.—*Case* 1. Part of the wall of the diverticulum shown in *Fig.* 255. It will be seen that the various layers of the bowel wall are all well represented. A, Villi and glands; B, Muscularis mucosæ; C, Muscular wall. It will be seen that the muscular wall is well represented, but the layers are irregular and thinned in comparison with the muscular wall of the bowel. (× 50.)

size—apparently about 1 cm. in diameter—the mucous membrane with the muscularis mucosæ herniates through between the muscle fibres of the main muscular coats. The important fact, however, remains that at the start the diverticula consist of all coats of the bowel. The significance of this in any consideration of the origin of the condition is discussed later.

The mucous membrane as it passes towards the apex of a diverticulum becomes thinner, as if from stretching, and the villi are fewer, shorter, and flattened. These changes are more marked in direct proportion to the size of the diverticulum.

In *Case* 5 a section of the wall of a diverticulum close to the area of perforation which had set up peritonitis shows hyperæmia and leucocytic infiltration, as might be expected, but this was the only evidence found of any inflammatory change in the specimens examined.

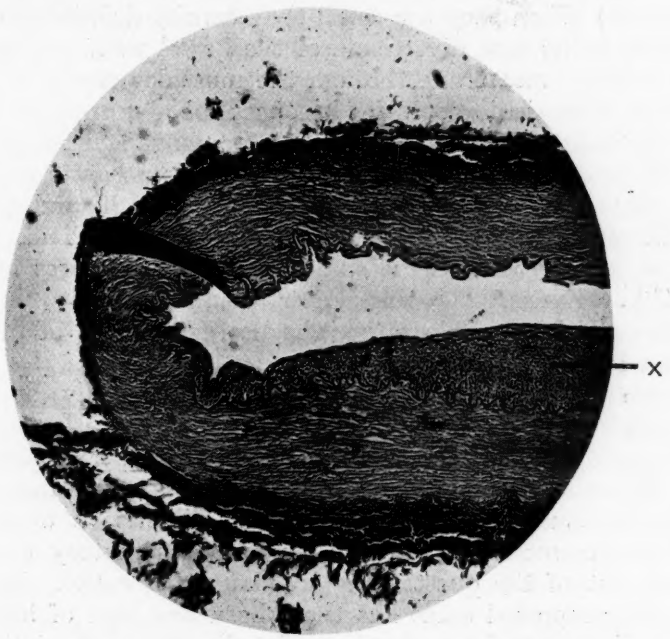

FIG. 257.—*Case* 1. Transverse section of one of the vasa recta running to the bowel wall opposite a large diverticulum. An area of very great subintimal thickening is seen at X.

Sections of mesenteric lymph-glands from *Cases* 3 and 6 show no abnormality.

Transverse sections were cut of the mesenteric vessels in each case, and particularly of the vasa recta approaching the bowel wall opposite the origins of diverticula.

In *Case* 1 subintimal thickening and narrowing of the lumen of the vessels is very marked in certain areas, though this is patchy, and other areas appear to have escaped (*Fig.* 257). *Case* 3 also shows areas of subintimal thickening and sclerosis of the walls.

Case 6 shows moderate sclerosis, but no marked areas of subintimal thickening in the sections examined.

In *Case* 5 only a very small amount of material for examination of the vessels could be obtained, as the mesentery had been cut close to the bowel wall, but the sections examined show no definite evidence of abnormality of the vessels.

ETIOLOGY.

In discussing the anatomy and the clinical manifestations of these diverticula, the following points have emerged and are important in any consideration of their causation: (1) These diverticula are not recorded in infants or young people; (2) They occur rather in persons elderly, or at least past middle age, and often present the appearance of being in the process of progressive formation; (3) Their points of origin at the mesenteric border of the bowel bear a very definite relationship to the vasa recta of the mesenteric vessels; (4) When they are small they consist definitely of all coats of the bowel wall, being true diverticula of the bowel wall, not hernial protrusions of the mucous membrane through the muscular coat.

That these diverticula could be a congenital abnormality, present from an early stage, is denied by the first two of the above statements. Moreover, they show no resemblance to congenital enterogenous cysts and diverticula. Such cysts contain all the elements of the bowel wall, and these are best explained as examples of heterotopia, or an isolation of islands of developing bowel wall at an early stage. A summary of several cases of the type is given by Arthur Evans,[7] but this writer would appear to include in this category some which resemble rather the particular type of multiple diverticula described in this paper.

Diverticula of the small intestine are occasionally associated with misplaced islands of pancreatic tissue.[16] No evidence of islands of pancreatic or other aberrant gland tissue can be found in the type we have under consideration. There is in fact no evidence that a congenital abnormality plays any part in the causation of this condition. Likewise a weakening of the bowel wall at the mesenteric border by an inflammatory or degenerative process seems out of the question as an underlying cause. In none of the writer's or other reported cases has there been any sign of inflammation of the bowel wall, apart from that associated with acute inflammation or perforation of a well-formed diverticulum.

Another theory of causation—that the mucous membrane herniates out along the sides of vessels passing through the bowel wall, as suggested by Grazer[9]—is denied by two facts. First, the diverticula at their commencement consist, as has been shown, of all coats of the bowel. Secondly, the only vessels with which they are in direct relationship at the start are the vasa recta of the superior mesenteric artery. These, as described by Cockinnis,[6] run alternately on to one or other side of the bowel, giving off branches at intervals, the first of which runs to the mesenteric border. The main trunk of the vessel does not penetrate the muscle coats at the mesenteric border, so that if penetration were necessary for the formation of the diverticula, these would be as likely to appear at the sides of the bowel as at the mesenteric border.

There remain two important possibilities: the presence of traction upon the bowel from without, and the presence of an increased intra-intestinal pressure acting from within.

Let us first consider the possibility of traction. The definite association of the diverticula with the points at which the vasa recta of the mesenteric arteries reach the bowel wall at once suggests that these vessels might, under certain conditions, exercise a pull upon the bowel wall at these points. Moreover, these diverticula appear first and are more marked in the first loop of the jejunum, the region where the vasa recta are longest and most fixed at their origins.

With a view to ascertaining the effect upon the bowel wall of exerting traction through the mesenteric arteries certain experimental work was carried out by the writer. Fresh post-mortem-room material was used, and in each case the first 12 in. of the presumably normal jejunum was taken and distended tensely with plaster-of-Paris cream. The arteries of the mesentery were also injected with red lead and plaster-of-Paris cream through the superior mesenteric artery.

As the bowel was distended the loop tended to straighten out, and when tensely distended the mesentery in the concavity of the loop formed a diaphragm pulled taut all round its circumference. The vasa recta of the superior mesenteric artery were therefore all put on the stretch. An X-ray of the specimen under these conditions shows the appearance of tiny diverticula opposite the point at which many of the vasa recta reach the bowel wall. The tiny diverticula consisted of all coats of the bowel wall and were therefore clearly small traction pockets. Further distension of the bowel resulted in the rupture of the muscle coats of these small pockets and herniation of mucous membrane, and in one case in bursting of the pocket with escape of plaster cream into the mesentery. An X-ray of a jejunal loop thus distended with plaster cream and with its mesenteric vessels injected, demonstrating these small traction pouches, is shown in *Fig.* 258.

Twelve experimental specimens were thus made, and it is perhaps of interest that the only two in which no pouches appeared were from individuals under 20 years of age, in whom the mesenteric vessels were probably more elastic than in the older cases. Moreover, the traction pouches were best marked in a specimen from a man of 65 with very marked generalized arteriosclerosis.

These experimental cases demonstrate that a pull exerted evenly through the mesenteric vessels in the presence of increased intra-intestinal pressure produces small traction pouches of the bowel wall very similar to the earliest stages of the multiple diverticula which have been described. It seems probable, therefore, that these two factors are also at work in the production of the multiple diverticula during life.

It seems quite possible that arteriosclerosis of the mesenteric vessels may be a very relevant factor. Not only would this lead to a loss of elasticity of the vessels, thus increasing the traction effect, but the narrowing of the vessels would lead to a decreased blood-supply to the bowel with consequent weakening of the muscle and a tendency to atonia and dilatation. Such dilatation of the bowel is seen at operation in advanced cases, and will be

likely to lead to a slowing up of the passage of bowel contents, both fluid and gaseous, and thereby to a rise in the intra-intestinal pressure.

In the writer's own four cases where tissue was available for microscopy arteriosclerosis of the mesenteric vessels was definitely present in three. In the fourth the vascular tissue available was insufficient for any definite statement to be made on this point as the mesentery had been cut close to the bowel wall.

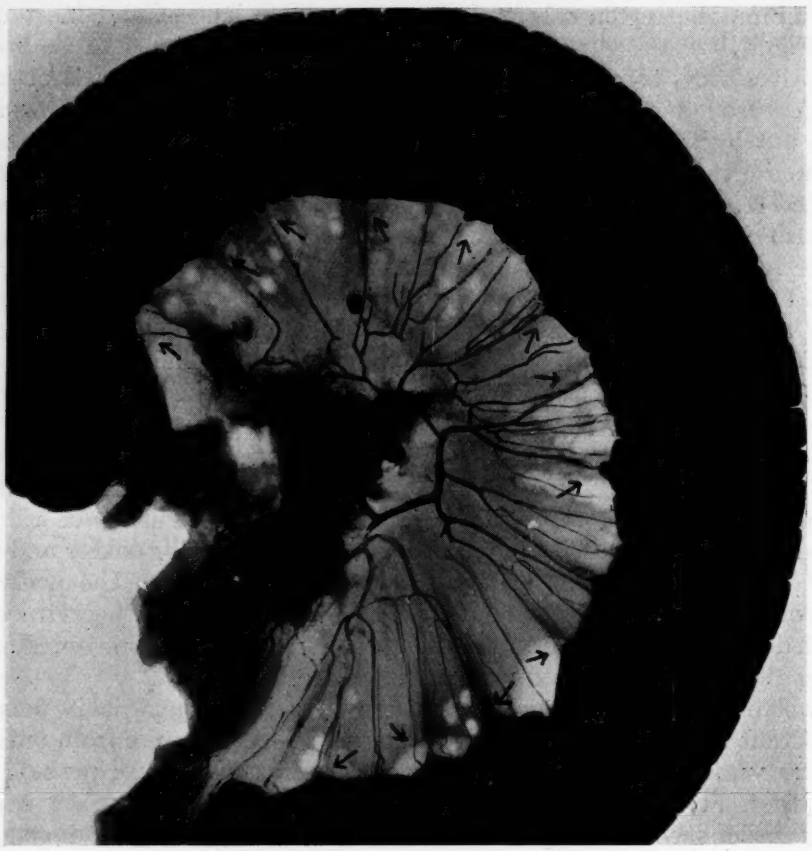

FIG. 258.—X-ray of first loop of jejunum from experimental case. The lumen of the bowel is filled with plaster-of-Paris cream. The mesenteric vessels are injected with red lead. Tiny traction pouches opposite the attachments of the vasa recta to the bowel wall are clearly visible. In the upper part of the picture the injected vessels showed somewhat faintly and have been emphasized for reproduction.

In so far, therefore, as the writer's own cases supply evidence mesenteric arteriosclerosis seems to be constant, and it would appear that it can act as the initial cause of both factors required to produce the condition—(1) traction by the vessels from without, (2) atonia of the bowel wall with consequent increased pressure from within.

Moreover, mesenteric arteriosclerosis as the initial cause of the condition fits in well with the clinical evidence obtained from an analysis of the histories of the twenty cases on which this paper is based. The average age was 64.

Arteriosclerosis was present to a degree sufficiently marked to attract attention of itself during life in nine of these.

In a total of 11 deaths, the causes were: cerebral degeneration 1, mesenteric thrombosis 1, coronary disease 1, uræmia 2, perforation of a diverticulum 2, pneumonia 3, acute obstruction 1. It will be seen that in the first five of these considerable vascular degeneration must have been present. In at least six of the twenty cases diverticula of the sigmoid were present, and in four chronic constipation was a marked symptom, and it is probable that these facts imply a degree of intra-intestinal pressure above the normal. Where the symptoms are found attributable to the fully developed condition, the most troublesome feature is often a feeling of distension, and there would seem to be no doubt that at this stage the intra-intestinal pressure is certainly raised as an almost constant condition.

Some degree of sclerosis of the mesenteric vessels must, however, be a fairly common occurrence in elderly people, especially those with marked general arteriosclerosis or chronic renal disease, but it is clear that all such cases do not develop diverticula. The explanation of this is probably that even though one factor, the increased traction, may be present, the other necessary factor, that of increased intra-intestinal pressure, fails to develop in many cases.

SUMMARY.

Multiple diverticula of the small intestine at its mesenteric border occur as an acquired condition, being found only in persons who are past middle age.

The writer suggests that the underlying causative factor in this condition is a sclerosis of the vasa recta of the superior mesenteric artery, which thereby exert an abnormal traction effect upon the bowel wall.

Small traction pockets are thus formed consisting of all coats of the bowel, which tend to grow to form large diverticula under any circumstances which increase the intra-intestinal pressure, whether such circumstances are due indirectly to the arteriosclerosis or to other causes.

Having reached a certain size, these diverticula grow much more rapidly, the mucous membrane herniating through the muscular coats.

In the early stages distinguishing clinical symptoms of the condition are usually absent, but in the advanced cases a definite set of symptoms may become manifest.

The condition may at times be diagnosed by X rays. The symptoms may in some cases be alleviated by medical treatment.

Radical surgical treatment is sometimes possible. Acute complications are rare, but occasionally occur, requiring urgent surgical aid.

The greater part of the work for this paper was carried out while the writer was House Surgeon to the Surgical Unit and Senior Surgical Casualty Officer at St. Thomas's Hospital. His sincere thanks are due to all those who allowed him to study cases under their care or placed material at his disposal

for pathological examination, particularly to Sir Cuthbert Wallace, Mr. C. R. Nitch, Dr. M. A. Cassidy, and Mr. B. W. Williams, of St. Thomas's Hospital; Dr. H. Nockolds and Mr. D. C. Taylor, of Lewisham Hospital; and Dr. W. G. Barnard, late Curator of the Museum of the Medical College, University College Hospital; also to Sir Arthur Keith for helpful advice and permission to study specimens in the Museum of the Royal College of Surgeons. His thanks are also due to Dr. Geoffrey Fildes, of St. Thomas's Hospital, for his great assistance in obtaining the clinical and experimental X-ray photographs.

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CLEFT PALATE.

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(*Being a Hunterian Lecture delivered before the Royal College of Surgeons of England on January* 27, 1933.)

The results of the treatment of cleft palate have left so much to be desired that a great many, disheartened at their efforts, have shunned the subject rather than admit their after-results. It is almost axiomatic that the lack of success in the treatment of any complaint is in direct proportion to the number of remedies, and the truth of this is amply illustrated in cleft palate by the number of methods which appears from time to time for its treatment. But from the morass of literature which has sprung up, one fact seems to be generally recognized—namely, that any treatment must aim at the restoration of contact between the posterior surface of the soft palate and the pharyngeal wall, whereby complete separation of the nasal and oral cavities can be effected. The ingenious method of Gillies and Fry was amongst the first devised with this aim.

THE MECHANISM OF SPEECH.

During normal speech, the palate mechanism is kept in a state of constant activity, the palatopharyngeal valve opening and closing at an extraordinarily high speed. Most of the consonants require closure of this valve, the exceptions being the *m*, *n*, and *ng*, where sound, and the air current, are allowed to escape through the nose. The remainder of the consonants are all explosives in that they require complete separation of the two cavities, so that air, voiced or unvoiced, should be pent up in the oropharyngeal cavity during or prior to the pronunciation. The *b*, for instance, requires 'voiced air' to be compressed before the final explosive effort which separates the lips and thus results in the production of the sound; *p* is made in the same way, with the exception that the pent-up air is not 'voiced' by the larynx. On the other hand, *m*, *n*, and *ng* require an open palatopharyngeal valve in order that the nasal resonance should be added. It will thus be admitted that destruction of the palatopharyngeal valve will place the possessor in an extraordinarily disadvantageous position in that the only sounds it is possible to produce correctly are the nasal resonants, as, of course, every attempt at the imprisonment of air for the explosive effort of the consonants is foiled by escape through the nasal cavities. The reverse of this is well seen in the common cold, where nasal obstruction converts the *m* into a *b*, the *n* into a *d*, and the *ng* into the hard *g* as in the well-known advertisement for 'Colbad's bustard' (Colman's mustard).

Actual closure of the palatopharyngeal valve is executed by two sets of muscles—those which move the palate and those which act on the pharynx. The muscles moving the palate are of two kinds—those which draw the palate to the pharyngeal wall and those which oppose this action and cause the palate to be drawn from the pharyngeal wall. Of the former, the levatores of the palate are the only ones of importance; they cause the soft palate to arise in a comma-like shape and make contact between its upper surface and the pharyngeal wall at about the level of the anterior arch of the atlas. Damage to, or destruction of, these levatores is the greatest disaster which can happen during any operation for the repair of the palate. The muscles causing withdrawal of the palate from the pharyngeal wall are those which enter into the pillars of the fauces—namely, the palatoglossus and the palatopharyngeus, and to a very small extent the tensor palati.

The other or pharyngeal half of the palatopharyngeal valve is formed by the upper part of the superior constrictor muscle of the pharynx, which, to effect closure of the valve, contracts strongly and produces a well-marked ridge or cushion on the posterior pharyngeal wall, at the same time narrowing the nasopharynx from side to side. This muscular action can be readily observed in any case of unrepaired cleft of the palate during the pronunciation of 'Ah' with the mouth wide open. It was first described by Passavant, and his name is usually attached to its description. In addition to causing this narrowing of the nasopharynx, the cushion of Passavant also causes the two halves of a cleft soft palate to be approximated, and recognition of this fact led me to prophesy the existence of fibres of the muscle passing into the soft palate and not terminating, as in the standard descriptions of the anatomical text-books, in bony or ligamentous structures. Dissections proved this prophecy to be correct and the observation was amply confirmed by my colleague, Dr. James Whillis, and has been more fully described by him in the *Journal of Anatomy*.[1] The importance of the observation lies in its proof that the superior constrictor is in fact a palatal muscle and therefore part of the palatopharyngeal sphincter mechanism.

DEFECTS IN THE NASOPHARYNX IN CLEFT PALATE.

In a previous Hunterian Lecture,[2] measurements taken by myself from a series of normal and cleft-palate skulls were quoted to show that in cleft palate there is a marked increase in the bony diameters of the nasopharynx, and that any operation designed to restore the palatopharyngeal valve must take account of this, otherwise the palate might be repaired with the best possible æsthetic result yet no closure of the palatopharyngeal valve attained. It was shown that the hamular processes which act as pulleys for the tensors of the palate were widely separated, and that if they remained intact the pull of the shortened muscle was sufficient to prevent suture without tension and with consequent immobility of the soft palate. Furthermore it was recognized that, the muscle being shortened, the effect of putting tension on the palate from side to side was to cause further shortening in an antero-posterior direction. To overcome this difficulty it was advised to destroy

the hamular processes.* But as all the diameters of the nasopharynx are increased, mere reduction in the tension from side to side is not likely to overcome this deficiency, and for this purpose the operation of pharyngoplasty was devised.

PHARYNGOPLASTY AND REPAIR OF THE PALATE.

This operation has for its object the imitation and exaggeration of the normal method of palatopharyngeal closure—that is, an artificial, but greatly exaggerated, cushion of Passavant is created on the posterior pharyngeal wall. The method of carrying it out is similar in principle to the Mickulicz pyloroplasty, with the difference, of course, that the operation is designed to narrow and not to widen the channel. Briefly, a transverse incision is made on the posterior pharyngeal wall and passes through the superior constrictor muscle. The incision is then sutured in a vertical direction, thus effecting reduction in the volume of the nasopharynx.

Anatomical Considerations.—The superior constrictor muscle lies immediately beneath the pharyngeal mucosa. Its posterior surface is closely enveloped in a thin but tough aponeurosis, the buccopharyngeal fascia, which separates the muscle from the loose connective tissue of the retropharyngeal space. It is extremely important to recognize this relationship, since the buccopharyngeal fascia protects the retropharyngeal space from perforation during the course of the operation. At each side of the nasopharynx a strong fold of fibromuscular tissue passes vertically downwards from the Eustachian tube and the base of the skull and encloses in its substance the salpingopharyngeus muscle. Recognition of this bundle is very important, since it must be included in the sutures used to narrow the nasopharynx. Deep to these salpingopharyngeal folds there is a fair amount of loose connective tissue, the buccopharyngeal fascia not investing the fibres of the superior constrictor so closely as on the posterior pharyngeal wall.

Technique of Pharyngoplasty.—

Anæsthesia.—The best anæsthesia for all palate work is endotracheal gas-oxygen and ether administered by the method invented by I. W. Magill. I would like to express my personal gratitude to Dr. Magill, who first demonstrated the method to me, and to Dr. W. J. Phillips, of Newcastle, for having taken the trouble to master the technique.

One great advantage of this method of anæsthesia is that bleeding and excretion of mucus do not interfere with the airway, and the operation can be carried out in a deliberate fashion without the constant aspiration which seems to occur with any of the other methods. Moreover, a long operation is suffered by even a young infant without any material deterioration in the general condition. At first the endotracheal tube was a nuisance, but with practice it can be tucked away to the right of the mouth at the side of the tongue.

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* Destruction of the hamular processes was, I believe, first advocated by Billroth. I was in complete ignorance of this when I arrived at the same conclusion as the result of my anatomical studies.

The Operation.—Using a headlight, the operation is carried out in a semi-darkened theatre. The patient is anæsthetized and the head thrown well back. A gag is placed in the mouth, the tongue being well depressed. By far the most useful gag is the Boyle-Davis as modified by Mr. Norman Dott, of Edinburgh. Mucus is swabbed away, using small gauze mops wrung out of 1–150 carbolic lotion.

The halves of the soft palate are held aside by small sharp Gillies' hooks and the nasopharynx is then lightly stimulated to produce reflex contraction of the pharyngeal muscle and to mark the level of the ridge of Passavant. Using a sharp-pointed knife, a transverse incision is made through the mucosa and the superficial fibres of the superior constrictor muscle at about the level of the ridge of Passavant. By means of a special dissector, really a standard aneurysm needle cut short, the constrictor muscle is gently peeled from the surface of the buccopharyngeal fascia in both an upward and downward direction until a considerable area is undermined. The peeling process is continued laterally right up to and beyond the salpingopharyngeal folds, and, using the dissector as a retractor, the incision is continued laterally up to these folds. During the process of dissection, care must be taken that the buccopharyngeal fascia is not perforated, since, although no trouble has arisen from this cause, it is not considered advisable to open up the retropharyngeal space with all its risk of infection. One can be certain of the fact that this space has not been perforated so long as resistance to the peeling process is encountered.

The business of suturing is now commenced, and using No. 0 catgut on a small Reverdin needle a suture is carried through the salpingopharyngeal fold on each side. It is extremely important to obtain a big bite of tissue and a firm hold, and on occasion the extreme edge of the upper surface of the soft palate has been included in this suture. The suture is not tied but is held with an artery forceps and acts as a very excellent retractor. Further sutures are then placed above and below the first until in all from three to five have been inserted. The remaining sutures, while used as a routine, are not felt to be of any great moment, but merely secure the coaptation of the edges of the mucous membrane. The sutures are now tied and cut short. It is impossible to make a really neat and tidy job of pharyngoplasty, but perhaps this is fortunate in that there is less likelihood of locking sepsis in a notoriously bad area. When the operation is completed it will be observed that the two halves of the soft palate lie much nearer the middle line and very often the halves of the uvula overlap.

There is no need to do any preliminary operation either for the removal of tonsils or adenoids, although the latter, if unduly prominent, may be a nuisance in that they interfere with the uppermost suture of the pharyngoplasty, or may give rise to some troublesome oozing of blood, thereby helping to obscure the field. The most important point to observe in this operation is the securing of a very firm hold of the salpingopharyngeal folds, since within a few days the sutures tend to soften and may come out before they have fulfilled their object. Difficulties are often encountered, particularly in infants, and mostly because of the small size of the parts and the extremely restricted area in which the operation is carried out. Once the

pharyngoplasty is completed, it behoves the anæsthetist to keep a sharp look-out for any sign of respiratory obstruction.

At this stage any further procedure can, if thought wise, be abandoned, but it is now my invariable practice to go straight ahead and repair the palate, since suture of the soft palate on top of a pharyngoplasty lends additional support to the latter, and at the point where the need is greatest —namely, at the insertion of the outermost sutures through the salpingo-pharyngeal folds. Moreover, the pharyngoplasty helps to reduce the tension on the sutures in the soft palate.

One criticism launched against this operation is the statement that a successful pharyngoplasty is not likely to remain in this condition, and that the artificial narrowing will disappear and the parts return to the normal state of affairs. Observation of many cases over a period of the last seven years has shown that this does not occur. Apart from what happens within a few days of operation, no widening or giving of the nasopharyngeal part of the operation has ever been observed.

Complications and Dangers of Pharyngoplasty.—In my original paper published in 1928 mention was made of certain dangers of the operation. The chief amongst these was the risk of retropharyngeal cellulitis, and reference was made to a case in which this might possibly have been a cause of death, although the post-mortem evidence still left one in considerable doubt. In all, the operation has been performed on seventy-two patients without any sign of recurrence of this trouble, and this in spite of the fact that it is believed that, owing to careless dissection, the retropharyngeal space has been opened on two or three occasions. Even so, this statement should not lead to the belief that such an accident will always go unpunished.

One risk which is ever present after this operation when combined with repair of the palate is that of asphyxiation during the course of recovery from the anæsthetic, and the more successfully the operation has been done the greater the risk. The aim of the procedure is to restore a palatopharyngeal valve, and in order to be successful it is necessary to over-correct, and a great many of these cases at the end of the operation are quite unable to breathe through the nose, necessitating continued traction on the tongue until recovery is complete. Latterly in using Magill's method of endotracheal anæsthesia, it has been our custom to leave the tube in the trachea until reflexes are fully restored.

Deafness was a further complication mentioned in my previous communication, but this has not turned out to be the bogy expected. Several cases have had temporary deafness which has cleared up in a few days; in no case has it remained permanent. My experience has been that sufferers from cleft palate, whether subjected to operation or not, are more liable to this trouble than the normal person—presumably owing to irregularities in the Eustachian tube or from its increased exposure to infection.

Repair of the Palate.—In repair of the palate, the method used by myself embraces what I believe to be the best in the Langenbeck-Fergusson and the Veau operations.

Using the left index finger the position of the hamular process is located, and beginning outside this point a lateral incision is made and carried forward

on to the hard palate close to the alveolar margin. This procedure is carried out even where the cleft is of the soft palate only. By means of blunt dissection the hamular process is defined, and is then completely detached from its base either by cutting it with strong scissors or wrenching it with an elevator. Careful palpation will make certain of the complete separation of the process. This procedure is then carried out on the opposite side, and it will be observed that immediately after successful destruction of the hamular processes the two halves of the soft palate fall much more closely together in the middle line, and also fall backwards towards the pharyngeal wall. No regrets need be felt at the apparently wanton destruction of bone, since it has as its main object the complete freeing of the shortened tendons of the tensors of the palate. The tensors undoubtedly occupy the key position in that they prevent movement of the palate and must be destroyed at any cost.

Inserting an elevator into the lateral incision, the mucoperiosteum of the hard palate is gradually stripped up from the bone; the stripping being carried forward as far as the anterior end of the cleft and the lateral incisions enlarged to the same extent. The separation is then carried out along the edge of the cleft and at the junction of the nasal and oral mucosæ. Then, passing an elevator either through the lateral incision or beneath the mucoperiosteum in the borders of the cleft, the soft palate is separated from the posterior edges of the hard palate. This is carried out entirely without further incision of the mucous membrane, and it is not necessary—indeed, it is regarded as highly inadvisable—to make any incision through the nasal mucosa at this point. The use of right-angled scissors as in the Langenbeck-Fergusson operation is mentioned only to be condemned, as it is believed that this act, which cannot be carried out with precision, is likely to cause damage to the levator muscles. Moreover, submucous separation means that less raw area is left to granulate, and the chances of infection are consequently materially reduced.

It should now be found that the two halves of the separated palate will meet in the middle line and will lie comfortably against the raw bone from which they have been separated. Should this not be so, the lateral incisions are extended further forward and then carried inwards to the cleft so that the whole of the mucoperiosteum of the hard palate is lifted up as a flap, as in the method of Veau. It will then be found that this flap can be slid inwards and sutured in the ideal position, lying snugly against the bone and without tension. One great advantage of raising flaps is that they can be turned backwards so that the greater part of the separation of the soft palate from the hard can be further assured under the guidance of the eye. Furthermore, the posterior palatine artery with its fascial covering can be loosened from its bony canal, thereby allowing a little extra inward movement of the palate. A great deal has been written about the necessity of preserving this vessel, and while I would not advocate its division as a routine procedure, on the occasions when I have done this by accident or design I have seen no ill results following. Sometimes it is essential to divide both vessels so as to bring the two halves of the palate together without tension, and often further freeing can be effected by dissection in those parts of the lateral incisions lying behind the fractured hamular processes.

To complete the repair of the palate, the nasal mucosa is separated from the upper surface of the bone and in the suitable case a vomerine flap can be turned aside as in the method advocated by Veau. My own experience has been that while I subscribe absolutely to the soundness of Veau's opinions on this procedure, I have found that the very cases in which I most wanted to carry out his methods have been the very ones in which it has not been completely possible. At any rate, wherever practicable, the nasal mucous membrane covering the upper surface of the hard palate is sutured in the middle line. This principle of suturing the nasal mucous membrane with fine catgut is carried right along the whole length of the hard and soft palates as far as the tip of the uvula, having first of all, of course, pared the edges of the soft palate. The operation is completed by using fine silkworm sutures for the under surface of the soft palate and the mucoperiosteum of the hard palate. The Veau 'suture musculaire' has not been used in my cases.

Mortality.—There seems to have been some hesitance on the part of general surgeons to adopt this operation, and on grounds believed to be associated with an expected mortality. In all I have done pharyngoplasty on 72 patients, with a total mortality of 3. The first case occurred amongst the earliest of the series, and death took place within thirty-six hours of operation; the other two cases were the direct result of the anæsthetic. It is worth while examining these cases in detail.

Case 8.—A boy, aged 4 years, who was submitted to pharyngoplasty without repair of the palate. During the course of the operation it was noted that the stripping of the superior constrictor muscle was carried out without resistance, and it was not recognized till too late that the retropharyngeal connective-tissue space had been opened. The operation was completed uneventfully, but the child died thirty-six hours later from what was believed to be acetonæmia, although at the autopsy a slight degree of retropharyngeal cellulitis was present and a very thin purulent track extended from the site of operation to the mediastinum. This case was only the eighth upon which the operation had been done and it caused very close examination of the operative procedure. Since that date the retropharyngeal space has been accidentally perforated on several occasions, but there has been no recurrence of the trouble.

Case 78.—A girl, aged 6 years, who had had diphtheria about eight months before operation. A preliminary tonsillectomy was done, and after waiting about six weeks, repair of the palate was undertaken. The anæsthetic was chloroform and ether mixture administered by a student anæsthetist. Pharyngoplasty was performed, and during subsequent repair of the palate it was observed that bleeding suddenly ceased, and the child died. This case was a great lesson in that it proved that anæsthesia in these cases is very difficult and can only be carried out in reasonable safety by anæsthetists of great skill and experience. The student in this particular case had given many anæsthetics and was in my opinion a born anæsthetist, but it was undoubtedly unfair on my part to ask him to undertake a case of this nature.

Case 53.—Aged 3 years. Endotracheal gas-oxygen and ether was given by the method of Magill, and pharyngoplasty and repair of the palate were done in one stage. The operation presented no unusual difficulties, and at its completion was regarded as entirely satisfactory. The pharynx was mopped dry and the endotracheal tube removed and the child returned to bed. A few minutes later an urgent message from the ward caused an immediate visit to be paid, and it was seen that the child was obviously asphyxiated. The airway seemed to be completely free, but it was certain that the child had drowned in its own secretions. Whether

these secretions were from the stomach and resulted from vomiting, or whether they had been retained behind the soft palate and only appeared on removing the patient to bed, was never satisfactorily explained, but the lesson learnt was one which has since proved extraordinarily useful; the anæsthetic tube is now never removed until the reflexes have returned and until it is certain that no secretions remain dammed up behind the soft palate.

Pharyngoplasty, with or without repair of the palate, has, then, in my hands given an avoidable mortality of 4·1 per cent, and the question arises whether the results justify this loss of life. Unhesitatingly I say 'Yes', and especially in view of the fact that the operation is new and, as yet, experimental. Cleft palate is a deplorable deformity for those who are unfortunate enough to possess it, and very much greater risks than those outlined above would not prevent me from advocating my present methods. The reaction of one's medical friends in this respect is most illuminating; when they are faced with the problem of cleft palate in their own offspring they say: "We appreciate the risks, but operate, operate early, take plenty of time, and make a sound surgical job."

Consideration of the three deaths quoted above shows that in no case was death due to inherent defects in the operation, but only to the occurrence of avoidable accidents. But apart altogether from the mortality of any operation, it will be agreed that it would be hardly justifiable to take the risk of carrying it out if the patient were to hang between life and death and only recover after a prolonged convalescence. This has not been my experience. It nearly always happens that after operation there is some reaction in the form of a rise in temperature, and on rare occasions the temporary enlargement of cervical glands has been noted. One sickly child, aged 13 months, gave reason for some anxiety for about a week; it was fevered, and the palate never showed any healthy reaction; most of the sutures ultimately gave way, but the child recovered. Delay in attempting operation in this case would have been advisable.

Latterly, the choice of the right time to operate on infants has been greatly facilitated by the skilful medical supervision of Dr. J. C. Spence and other members of the staff of the Newcastle-upon-Tyne Babies Hospital.

THE RESULTS OF OPERATION.

The results of a new operation merit very careful study before a strict valuation can be placed upon them. First of all it is necessary to recognize that from a surgical standpoint all that can be obtained is the formation of a functional valve separating the oral and nasal cavities. No operation has yet been devised which will make the possessor of such a valve either speak properly or use it for the other functions for which it is required. In any discussion of the results, therefore, it necessarily follows that there are two criteria of success: first, the restoration of the palatopharyngeal valve; and, secondly, the restoration of normal speech. If the first criterion is attained, it does not necessarily happen that the second will also be attained, but it is quite certain that if the first criterion, the formation of a competant palatopharyngeal valve, is not attained, then the second, of normal speech, cannot possibly follow.

Restoration of the Palatopharyngeal Valve.—The question arises of how one is able to prove the existence of a competent palatopharyngeal valve. Peroral inspection of the valve is almost useless, and is a very unreliable guide as to what is taking place behind, since contact between the palate and the pharyngeal wall occurs at the level of the anterior arch of the atlas and at a considerably higher level than the uvula. This was forcibly brought home to me in one of my cases where it would have appeared from inspection that closure of the pharyngeal valve might be impossible, yet the patient was able to present proof positive on trying the various tests of function. Equally, other cases have been seen in which it would appear certain that the valve was competent, yet on further trial it was proved without doubt that palatopharyngeal closure was not being effected.

Fortunately there are a number of simple tests which are of very considerable importance and if applied properly are believed to be accurate; they can easily be carried out in any consulting-room. Of these the 'snorting test' is by far the simplest. The patient is instructed to put the tongue slightly out and grip it between the lips. Air is then sucked into the nose as in the impolite act of clearing away pharyngeal mucus. A characteristic sound closely resembling the snore of a deep sleeper is produced and can be sustained and repeated if pharyngeal closure is being attained. The reason for protruding the tongue and closing the lips is to prevent accessory noises being formed behind the tongue. Various degrees of snorting are recognized, and one fallacy is seen in some cases where closure can be maintained for just a fraction of a second, the valve only being competent with severe muscular effort. It is interesting to observe that snorting is sometimes obtained in cases where the soft palate is almost completely immobile, and presumably is due to the contraction of the pharyngeal muscle and possibly also to the slight pull of the levatores. Snorting is a natural attainment of the normal individual and is seen at its best in the presence of a chronic pharyngitis. It is an impossible feat in the case of the unrepaired cleft palate, a fact often reflected in the dirty state of the pharyngeal mucosa in such cases. It is equally impossible in the large majority of cases submitted to operation by the classical methods, and casts a great reflection on their results. As a means of assessing the efficacy of operation on the palate it is of inestimable importance. Holes in the hard palate do not alter its value, since the tongue effectively seals them and prevents air entry by any other way than through the nasopharynx.

A rough and somewhat inefficient test which can be carried out even in the presence of an incompletely repaired hard palate is the ability of the patient to blow out a match or a candle. Improvement in performance can be measured by the increased distance at which this is possible. A great fallacy is, of course, the degree of nasal obstruction which might exist and give a false impression of palatal closure.

Rubber balloons are very popular with children, and usually require such a degree of expiratory effort to inflate them that the feat becomes impossible if holes exist in the palate or if the nasopharyngeal valve is incompetent. As a test of strength it is excellent. One child, however, succeeded in evading the test by pursing the lips to such an extent that they overlay the nostrils

and so prevented nasal escape. A second child succeeded in inflating a balloon by using a mouthful of air at a time, the air being imprisoned between the tongue and the roof of the mouth and forced into the balloon by the pressure of the tongue, the lips being tightly closed on the rubber outlet between each effort. In the absence of these tricks, however, the test is reliable.

A further test is by the use of the nasal irrigator or aqueous spray, and is only applicable to adults or older children. The head is thrown back and a bland fluid (saline or weak antiseptic lotion) is introduced into one nostril until it begins to return through the other, a feat easily accomplished in the normal person. If the nasopharyngeal valve is incompetent, a reflex disturbance of a major sort is induced and the experiment terminates abruptly; consequently this test can only be classified as confirmatory.

Another possible test consists of connecting an airway between one nostril of the patient and the ear of the examiner. The apparatus consists of a rubber tube having at one end a glass nozzle to fit the nostril of the patient and at the other the ear-piece of a stethoscope. The patient is then instructed to say any of the consonants, with the exception, of course, of the nasal resonants (*m*, *n*, or *ng*). Nasal escape is accurately detected by the ear of the examiner. For this idea I am indebted to Sir Richard Paget.[3]

A more complicated method was demonstrated by me at the Royal Society of Medicine in June, 1927. Separate tubes are introduced into the mouth and the nostrils, the latter being Y-shaped and completely filling both nostrils. The nasal and oral escapes are collected in separate gas-bags and can be measured. In use, the patient is instructed to blow through the mouth tube without allowing nasal escape, an impossible feat if the palatopharyngeal valve is incompetent. The amount of air collected in each bag is then measured and the degree of incompetence can be estimated. By a simple mathematical calculation the cross-section area of pharyngeal deficiency can be arrived at, although it is admitted that there is a very large degree of experimental error.

So much for the surgical problem of the formation of a functional valve and the proofs of success. Before passing to the second criterion of restoration of normal speech further important factors require consideration, and particularly those responsible for the production of cleft-palate speech.

Causes of Cleft-palate Speech.—A child born with a cleft palate wakens to a sense of its deficiencies in the first few hours of life when it realizes that the business of feeding is not so easy as it might be. In the complete cleft, sucking the breast or the bottle is an impossible feat unless the nostrils are at the same time occluded, and deglutition and aspiration must at first be extremely difficult to distinguish. These problems surmounted, the business of speech is the next in order, and the child's first effort to communicate with its fellows—namely by crying—offers no serious difficulty. The first word uttered by most infants is 'Mamma', and as the consonants of the word are nasal resonants and require an open palatopharyngeal valve this is easily accomplished. But when the second word 'Dadda' or 'Gagga' is attempted, the incompetence of the nasopharynx becomes a real problem. If the hearing

is acute and the speech sense good, the cleft-palate infant will recognize that there is only one way of saying 'Dadda'—namely, by placing the tip of the tongue against the roof of the mouth immediately behind the alveolar margin and then allowing the explosive effort to take place as the tongue is suddenly withdrawn. If this realization of the physical disabilities of speech is clearly brought home, the child will grow up using the tongue and palate, making the consonants in the correct way and in spite of nasal escape. The task will be rendered the easier the less the degree of the cleft, but it does not necessarily follow that the less the degree of the cleft, the better will be the speech.

Such cases as those outlined above are not uncommon in cleft-palate practice and offer by far the best type on which to attempt operation, and for purposes of classification have been labelled *Group* 1.

Let us imagine a second group of cleft-palate speakers. In *Group* 2 the child has a fair degree of acuity of hearing and is a trier. It attempts the big stumbling-block, 'Dadda', and finds that nasal escape prevents reasonable expression. It looks round for a means of overcoming this nasal escape and finds that movement of the muscles controlling the alæ nasi assist in preventing this. Herewith is born one of the most characteristic of the articulatory gymnastics of the cleft-palate speaker, and one which very often remains long after the palate has been successfully repaired, but fortunately does not interfere materially with speech training. It takes the form of violent occlusive movements of the alæ of the nostrils during speech, but although it may assist in reducing nasal escape it does not succeed in removing the stigma of cleft palate. This observation can be easily verified by nipping the nostrils of any cleft-palate speaker, when it will be observed that the type of speech does not alter although it may be somewhat modified. Such a child will often have difficulty with the *s*, and discovers that this sound, instead of being made between the tongue and the teeth, can be formed in quite a reasonable way in the pharynx, and thus is born another of the characteristic cleft-palate sounds which give so much trouble in re-education. The sound is not always easy to recognize, as it can be made in such a skilful fashion that it resembles fairly closely the normal *s* sound. Its recognition in cases of doubt is, however, made simple because the cleft-palate *s* can be made with the mouth wide open, a feat which is impossible in the normal *s*. The consonants, with the exception of the nasal resonants (*m*, *n*, *ng*), require a closed nasopharyngeal valve and in all the oral air-stream is partially or completely interrupted in some part of their formation, and it is at this point that the glottic stop is introduced as a means of disguising the absence of adequate oral interruption. It is used by completely closing the glottis and placing tongue, teeth, lips, etc., in their more or less correct relations and then suddenly releasing the glottis to allow of the escape of air and the formation of the sound. In this way a very fair imitation of many of the consonants is possible, but such speakers are mostly difficult to understand. (The glottic stop is occasionally used in the English language where two vowel sounds meet as in 'the ether'.)

In *Group* 3, or worst group, where one presumes that acuity of hearing and intelligence are at their lowest, it may be that not even the trick of the

glottic stop is acquired and all the sounds resemble those of vowels. Such speech, except to the ears of love as represented in the patient's nearest and dearest, are completely unintelligible. But it is not fair perhaps to think that all such are untrainable.

Consideration of the above factors in the causation of cleft-palate speech provides the clue to the reason for the failure after any operation however carefully performed. If the child is of the type mentioned in the first group, where the speech mechanism is brought into correct anatomical position for the formation of each sound, then the supply of an adequate or even slightly incompetent palatopharyngeal valve can be expected to result in perfect speech, or in speech that is nearly perfect. If the cleft-palate gymnastics have been thoroughly acquired, no operation of any sort can possibly help without speech training in which these tricks are unlearned and replaced by normal methods. It is useless, therefore, to classify cases according to the speech results alone, since these merely indicate the proportion of ambitious patients and the skill of the speech trainer. Wherefore in the consideration of the results of operation my cases have been divided into four main groups :—

1. Firstly, those who possess the prime factor of importance—namely, a competent palatopharyngeal valve—and who therefore have the functional physiological mechanism for the production of normal speech ;

2. Secondly, those who speak without any cleft-palate stigma and are therefore, apart from their dialect and other peculiarities, perfect speakers ;

3. Thirdly, those who have neither normal speech nor a physiologically competent valve ; and

4. Fourthly, those who are too young to apply the various tests or to make certain of their condition by their speech ; also cases in which the operation is of recent date.

As mentioned previously, the operation of pharyngoplasty has been done on 72 patients ; 3 of these died ; 3 have not been traced ; in 10 the operation was done as a secondary procedure and will be considered later ; and in one case, although the pharyngoplasty was done about two years ago, repair of the palate has not yet been completed. This leaves 55 patients for consideration.

Discussion of Results shown in Table I.—The cases recorded above have not been chosen in any way either as to age or type. Many had had previous operations by other surgeons. They represent all-comers, and I do not recall ever having refused to operate excepting in one case with diabetes. As will be observed, the age of operation varies between 6 months and 45 years, and can be accepted as the usual run of an average surgical practice. It will be noticed, however, that only three of the cases classified as possessing competent valves have been submitted to operation before the age of fluent speech, whereas amongst the group of perfect speakers are only three who had passed this age.

Some of these cases are worth considering in further detail in relation to their respective groups.

Taking first those where valves have been successfully formed : *Cases* 16, 24, 28, 62, 79, and 5 all possess excellent palatopharyngeal valves, and

although they all belonged to the *Group* 2 of cleft-palate speakers, as a result of training, either professionally or by their parents, they are able to obtain every sound in normal speech and to pronounce each sound without any fault whatever, yet they cannot be transferred to the group of perfect speakers, since, if they are taken unawares, the speech immediately develops the characteristic stigmata. In none of my cases is this allowed to pass as perfect speech, although I feel that I should be justified, since obviously the valve mechanism has been sucessfully restored and moreover the patient can speak perfectly at will.

Table I.—Results in Fifty-five Cases of Cleft Palate.

Competent Valves			Normal Speech			Defective Speech and No Valve			Too Young to Ascertain or Operation too Recent		
Initials	Case No.	Age at Op.	Initials	Case No.	Age at Op.	Initials	Case No.	Age at Op.	Initials	Case No.	Age at Op.
		Years			Months			Years			Months
L. G.	75	3	B. S.	34	6	G. W.	15	$2\frac{1}{12}$	J. R.	47	11
L. G.	60	$3\frac{1}{2}$	E. S.	31	7	J. A.	76	11	J. W.	67	11
W. G.	71	$3\frac{3}{4}$	J. B.	29	11	W. P.	1	17	A. G.	58	12
H. B.	69	6	H. H.	57	25	A. E.	41	19	D. H.	21	13
A. S.	62	7	E. B.	14	26	S. C.	2	24	J. W.	27	14
T. W.	20	7	J. D.	59	27	D. S.	9	45	C. P.	45	14
J. B.	11	8	J. L.	52	31				A. R.	93	21
J. W.	19	8	V. D.	48	34						Years
N. M.	7	9			Years				M. D.	98	2
T. B.	12	10	J. T.	13	3				J. P.	66	2
R. S.	79	13	M. T.	40	$3\frac{3}{4}$				J. B.	89	2
G. M.	3	13	P. Mc.	22	7				H. M.	37	$2\frac{1}{2}$
E. F.	6	15	J. J.	18	23				J. G.	68	$2\frac{1}{2}$
G. W.	82	17	E. S.	80	28				J. A.	91	$2\frac{1}{2}$
W. K.	16	18							E. S.	83	3
Mrs. S.	5	26							O. N.	84	3
E. F.	24	32							N. S.	61	7
G. B.	28	34							A. McQ	99	18
D. S.	4	36									

Case 75 was taken to the Antipodes before the end-result of this operation was seen by myself; he was able to snort in my presence, proving his possession of a valve, and shortly after his operation his mother stated that he could say many words with perfect clearness, although this latter was not sufficiently demonstrated to me. It is possible that he may by now, after the lapse of four years, be able to speak perfectly.

Case 20 presented a most unusual state of affairs. He had a cleft of the hard and soft palates, and was submitted to pharyngoplasty followed by repair of the palate. The palate broke down, and two further attempts resulted in failure in that the soft palate gave way along almost its entire length with the exception of about half an inch at the posterior extremity. Abject despair of ever succeeding in this case was only natural, but the boy turned up some twelve months later with the enormous holes completely closed and a freely mobile palate. Moreover he can blow up a balloon—for a child the hardest of all functional tests; speech has improved immeasurably;

the remaining defects are slight, and he is young enough to have these remedied by a short course of training.

In the second class—namely, the perfect speakers—are many cases that are worthy of note :—

Cases 13, 14, 29, 31, 34, 40, 48, 52, 57, and 59, were all submitted to operation either before speech had begun or shortly afterwards. In every case the only speech training has been given by the mother, and of course one finds that mothers vary immensely in their capabilities in this direction. It has been very profitable to me to see these cases at fairly frequent intervals and observe the development of the speech habits. Many of them have had baby talk which has been obviously cleft-palate in type, but almost invariably they have, early in their careers, shown evidence of the presence of a palato-pharyngeal valve in a curious sort of snore executed at intervals whilst attempting to form words. Gradually the snore has disappeared and the stigmata of cleft-palate speech have disappeared with it, until the only remaining fault has been with the double consonants, which appear to give exceptional difficulty. In the course of time even this has been remedied, until now no defect in speech is observable. Some of these cases have not yet got over their baby talk, but their loquacity has enabled one to be certain that there is no stigma of cleft palate, nor is such likely to develop.

Case 18, a woman aged 23 years, had a cleft of the soft palate which reached, but did not involve, the hard palate ; she was a *Group* 1 speaker and formed all her consonants in the correct fashion but was extremely difficult to understand because of the gross nasal escape. Pharyngoplasty and repair of the palate were done in one stage. After removing the sutures about the tenth day, the patient was made to read from a book, and with a little correction at first she was able to make all her sounds perfectly. She could snort strongly from the very first. This young woman is now, three years after operation, a perfect speaker and does not present the slightest stigma of cleft palate. She has had no training beyond the demonstration given after removal of the sutures.

Case 80, a woman aged 28 years, had a cleft of the soft palate almost reaching the hard. Before operation she spoke with gross nasal escape but did not possess any of the usual articulatory faults, and therefore belonged to *Group* 1. Her operation was performed to enable her to earn a livelihood in Australia and it was expected that she would leave within a week or two of being discharged from hospital. Little interest was therefore shown in her after-progress, but to my surprise she came to see me four years later, and not knowing her again, owing presumably to changes in female fashions of dress, it was only after protracted conversation that I was able to ascertain that she had come to show off her palate. She had not had training, but her speech was just that of any ordinary person. She was unable to inflate a balloon, but could snort in a most convincing manner.

Case 22 is the most remarkable result in the whole series and is recorded in the section dealing with prognosis.

Amongst the bad results are recorded cases which are still under treatment but in which no evidence has yet been adduced to show either a palato-pharyngeal valve or perfect speech, although I am sanguine of effecting at

least the formation of the valve in all. *Case* 41 is a mentally dull girl aged 19 years, who had a very wide cleft of both hard and soft palates with little tissue to spare. Operation was most successful in that the cleft was completely closed and the soft palate freely mobile, but no evidence of the restoration of the valve has yet been produced. *Cases* 1, 2, and 9 were amongst the earliest of my series and are recorded in a previous Hunterian Lecture.[2]

'Too young to ascertain' results include a number of cases where operation has been performed in the last few months or where the child has not yet spoken sufficiently to warrant the speech being classified either as normal or definitely cleft-palate in type. *Cases* 27, 45, and 60 speak but little, some of the sounds being normal in character and others of the cleft-palate type; but in view of previous experience with regard to the development of speech in other children of this age, it is not felt possible to assign them to any other group, although there is some evidence that they are capable of palato-pharyngeal closure.

In *Table II* the cases are classified according to the type of cleft, and it will be noted that most of my cases have involved both soft and hard palates or have been complete unilateral clefts with hare-lip.

Table II.—Type of Cleft in Fifty-five Cleft-palate Cases.

Result	Soft Palate		Soft and Hard Palate		Unilateral Complete		Bilateral	
	Initials	Case No.	Initials	Case No.	Initials	Case No.	Initials	Case No.
Competent valves	G. M.	3	N. M.	7	L. G.	75	J. B.	11
			T. W.	20	H. B.	69	W. G.	71
			R. S.	79	A. S.	62		
			E. F.	6	T. B.	12		
			E. F.	24	W. K.	16		
			J. W.	19	Mrs. S.	5		
					G. B.	28		
					D. S.	4		
					L. G.	60		
					G. W.	82		
Normal speech	J. T.	13	B. S.	34	H. H.	57		
	M. T.	40	E. S.	31	E. B.	14		
	J. J.	18	V. D.	48	J. D.	59		
	E. S.	80	P. Mc.	22	J. L.	52		
			J. B.	29				
Defective speech and no valve			W. P.	1			G. W.	15
			W. E.	41			J. A.	76
			S. C.	2				
			D. S.	9				
Too young to ascertain result or operation too recent	J. W.	67	J. W.	27	D. H.	21		
	J. B.	89	J. R.	47	A. G.	58		
	A. R.	93	C. P.	45	N. S.	61		
			H. M.	37	J. G.	68		
			J. P.	66	O. N.	84		
			E. S.	83	J. A.	91		
			M. D.	93				
			A. McQ.	99				

PROGNOSIS AFTER OPERATION FOR CLEFT PALATE.

The prognosis after operation depends on many factors, the first being the age at which the operation is done—that is, whether done before or after speech is developed. It can be said that where the operation is done during infancy, and *where a functional palatopharyngeal valve has been formed*, speech will develop along normal lines without any training. It should be the aim of every surgeon who is dealing with these cases to put the speech trainer out of work, and the only way to do this is by operation before faulty speech habits are formed. In an earlier part of this paper mention was made of the imaginary attempts of the sufferer from cleft palate to overcome his lack of a palatopharyngeal sphincter, and the importance of operation at this stage, before the development of articulatory gymnastics, cannot be overstressed. Cleft palate is a deformity which should be treated as a surgical emergency, and should be submitted to operation as soon as the health of the child and the skill of the surgeon will allow. Operation is often extremely difficult, but rarely dangerous, and requires patience, courage, and skill with sometimes but little reward. Nevertheless such deterrents should not weigh in the attempt to obtain better results in this most distressing complaint.

When the sufferer from cleft palate has reached that stage at which speech has been acquired, the whole question of prognosis takes on an entirely different aspect, and within limits this aspect is not materially altered by the anatomical defect present. Apart from the surgical difficulties, the main factor to be taken into account is the character of the speech acquired. As was mentioned previously, cleft-palate speakers divide themselves into three rough groups, and it stands to reason that in given anatomical circumstances operation on these three groups will produce widely different results. If all are provided with a properly functional mechanism, although the result of the operation is the same, the speech will deteriorate in order from the first to the third group. A great deal of literature has been produced on the speech results of operation for cleft palate, but in none that has come under my notice is this, the most important feature, noted, and it would seem to be obvious that no record of results after the age of speech is of the slightest use unless the speech before operation is taken into account. It is worth while examining these three groups in detail, *assuming that as the result of surgical treatment each has been provided with a functional palatal mechanism.*

Group 1.—In the first group the consonants are formed in the correct way but with nasal escape. In every such case it is almost certain that normal speech will be restored after operation, and this may be done without any training whatever, the only stigma likely to remain for some time being the 'nasal grimaces' which are so characteristic of the habit acquired while attempting to prevent nasal escape. In the reasonably intelligent patient a few lessons demonstrating the use of the palatopharyngeal valve may be necessary, and this may be all that is required. Two of my cases (*Cases* 18 and 80) fell into this group and are recorded above.

Group 2.—In the second group is included the greater part of the cases in the average surgical practice. The patients have acquired faulty speech

technique which may render them almost unintelligible to the uninitiated listener. They are the quintessence of joy to the speech trainer, since they provide his livelihood for many a long day. The habits of a lifetime have to be unlearned and new habits acquired, a feat which becomes increasingly difficult as age advances. Of all the cases of cleft palate these are undoubtedly the worst to manage, because, in spite of an operation which should be regarded as highly successful in its anatomical and physiological result, speech may improve but little. Acuity of hearing, a certain amount of intelligence, and, above all, ambition, are necessary for complete success, and up to the present these factors are entirely beyond the control of surgery. It is sometimes marvellous to behold the good influence of an approaching courtship or marriage, and sometimes also to observe the return of carelessness after the desires are attained. Amongst my greatest disillusionments in cleft-palate surgery was the case of a young woman who belonged to this group (*Case* 5) and upon whom the operation of pharyngoplasty with repair of the palate was performed. The operation resulted in a perfect palato-pharyngeal mechanism which complied with all the tests, and for many weeks I spent much time in correcting her bad habits, until in the end she was sound perfect. At this time she was engaged to be married, and, marriage ultimately taking place, I did not see her till some months after, when, happening to meet her in the street, I was amazed to find that she had become one of the worst cleft-palate speakers I had ever heard.

It must surely, then, be realized that while surgery is able to provide a perfect speech mechanism, and speech training in the suitable case can complete the cure, there is no operation which will make the patient use the mechanism and the training in the ordinary duties of life. This case is amongst my best results, yet remains amongst the worst speakers, and it is hardly fair to include it in the surgical failures, but it certainly supports the plea put forward to regard the cleft palate of infancy as an emergency.

In this and the next group there is a certain proportion of the patients who are quite untrainable from lack of ambition, self-consciousness, or sheer crass stupidity, and it is felt that it is equally unfair to attribute this to any operative procedure, since they may possess the mechanism, but for the reasons mentioned above are quite incapable of using it. There is much to be said for the method of Ernst, who, in dealing with cases after the speech has been acquired, first of all fits them with an obturator and attempts to train them in speech; if they are trainable, he proceeds to operate.

Group 3.—The third group contains all those who are incapable of forming consonants at all, and who are consequently unintelligible. The glottic stop is usually freely used and serves to separate the vowels from one another, and in this way a form of speech is acquired. It is wrong to assume that such cases are lacking in intelligence, acuity of hearing, or any other abstract quality, as the following will show. A boy of 6 years was brought to me with speech of this type (*Case* 22). He had a cleft of the soft palate which also involved about half the hard palate. Speech was quite unintelligible. Pharyngoplasty was performed, and fourteen days later the palate

was repaired by the Langenbeck-Fergusson method. Within three weeks of the second operation he was able to use his palatopharyngeal valve and could blow soap bubbles. Within three months all his consonants, the result of speech training, were perfect. He now speaks perfectly and without the slightest stigma of cleft palate, although he possesses a broad Tyneside accent. This boy has been lucky in that he has never acquired thoroughly bad speech habits, and the only trick he has had to unlearn is the glottic stop. He is an intelligent little lad and gives the lie to those who suggest that all patients in this group of speech are all mentally deficient. That some may be mentally dull is true, but I have the feeling that I would rather tackle some cases in *Group* 3 than a great many of those in the previous group.

SPEECH TRAINING.

Most of the literature dealing with cleft palate finishes in pious vein with the suggestion that an essential feature in treatment consists of training after operation, and there the matter ends. My experience of cleft palate has taught me that if successful results are to be obtained there must be close co-operation between the operator and the trainer. One cannot get on without the other, each working in a little watertight compartment, and if they are to co-operate with useful effect it is necessary that the trainer should understand the essential surgical and physiological requirements of the operative treatment, and for this purpose there is no better educational exercise than the attendance at a few operations.

Speech training is not only something to be used after operation where the result is obviously bad. It can be used to some purpose before operation by an intelligent mother where a child with cleft palate is first beginning to talk, and if she is successful she will assure that the operation will restore normal speech. During this time of life it is most important that the child should be taught correct habits in that the tongue, lips, and teeth should be placed in their correct positions for the formation of any given sound. Where a child is obliged to await operation it is my custom to instruct the mother either not to encourage the child to talk at all, or to see that if it does talk the sounds are formed in as perfect a manner as possible.

But there is no doubt that it is after operation that speech training finds its greatest sphere of usefulness, and its method of application depends on whether the child has been operated upon before or after the talking age.

If the operation has been completed before the child talks, the training as such rests, as it does in most of the population, in the hands of the mother, and, apart altogether from cleft palate, the mothers of this world are the greatest speech trainers at all times. But much can be done to help at this stage, and the first essential is to make the child 'valve conscious' by means of any form of blowing exercise which for its successful execution requires closure of the palatopharyngeal valve. Trumpets, squeakers, soap bubbles, and balloons are popular toys with children and can be made to exercise an immense influence on the subsequent course of speech. It has been my experience to observe on many occasions the improvement which takes place when blowing exercises are religiously carried out, and many children in

whom the baby talk is obviously cleft-palate in type are restored to normal speech within a few months. It was very interesting to observe one child who improved in speech until everything was perfect with the exception of some of the double consonants—for instance, the *s* as in 'swim'. In the course of a few months even this defect was completely corrected—a great tribute to the value of operation at or before the age of experimental speech.

Where the operation has been completed after speech has commenced, the training becomes a much more complicated process, and resolves itself into the correction of faults as well as instruction in the proper method of forming sounds. However, it is advantageous to discuss this question in relation to the various groups described earlier on. To recapitulate: *Group* 1 consists of those whose only fault is nasal escape; *Group* 2 have learnt all the articulatory faults; *Group* 3 are quite unable to form any consonants but use vowels only, with or without the glottic stop.

Group 1 speakers have only to be taught the use of the palatopharyngeal valve, and gain very material help from blowing exercises or from learning to play some wind instrument. Sometimes they have difficulty at first with blowing, and can often be helped by being instructed to snort several times and then to blow, keeping the soft palate in the same position as during the snort. It is not often that failure follows a demonstration of this kind. There is only a short jump between the successful blowing exercises and the perfect formation of the consonants without nasal escape, since, where a defect is found to exist, snorting followed by a repetition of the consonant is usually effective, and has proved an immensely good method in many of my cases. This is really all that is required with the *Group* 1 patient, and my experience has been that perfect speech follows as a natural sequel. Bad habits can be controlled from time to time by the use of a small mirror placed horizontally and glass side up beneath the nostrils, when any nasal escape is signified by steaming of the glass.

Group 2 cases present the greatest problem in that their faults have to be remedied and replaced by normal methods. There are three main faults in this type of speech: (1) Nasal escape; (2) Complete absence of a consonant and its replacement by the glottic stop; and (3) Formation of the sound in the wrong position. Nasal escape can be corrected by the method outlined above for *Group* 1 cases. Absence of the consonant and its replacement by the glottic stop is one of the commonest faults and is very difficult to eradicate, since, as a habit, it is usually well ingrained. The consonants affected by this method of speech are mostly the *t*, *d*, *k*, *g* (hard), and any words containing these letters, at any rate in their middle or at the end, are simply slurred over. In most cases the tongue is placed within reach of the correct position, but instead of interrupting the air-stream in the mouth it is interrupted in the glottis, an illustration of the misuse of the glottic stop. The word 'better' thus becomes 'be/er', 'adder' becomes 'a/er', 'sparkle' becomes 'spar/le', and so forth, the stroke in each case representing the glottic stop. Particularly obnoxious is this habit in words terminating in these consonants, as in 'but', which becomes 'bu/'.

In restoring the *k*, *g*, *t*, and *d*, it is best to start with the voiceless sounds

and demonstrate the position in which it is necessary to place the tongue in relation to the hard palate and attempt to form the explosive effort as 'kuh', 'tuh', rather than uniting consonants with a vowel as in 'kay' or 'tee'. Sometimes nipping the nostrils to prevent nasal escape assists materially in this. The keen patient rapidly acquires this feat and one may then proceed to introduce vowel sounds, giving as exercise 'kar, kay, key, ko, koo', and also 'ack, ache, eke, ike, oak, ook', etc. From this the voiced consonants are then introduced, and finally double consonants.

Formation of the consonant in the incorrect position is best illustrated by the cleft-palate *s*, where the sound is made somewhere in the pharynx, the exact site of which I have never been able to locate accurately. This sound can be made with the mouth wide open, a feat impossible for the normal speaker, and although I have learned to mimic the cleft-palate speech with a considerable amount of success, I have never been able to reproduce the cleft-palate *s*. The correction of the faulty *s* is not really difficult, and can be done by instructing the patient to place the tongue in position as though about to say *t*, but, instead of making the explosive effort, to allow the air to escape between the tip of the tongue and the anterior end of the hard palate immediately behind the alveolus, the trainer meanwhile nipping the nose if necessary. One of my cases after learning the correct method was unable after the lapse of about a fortnight to repeat for me the cleft-palate *s* which had been a life-long habit. Having learnt the correct *s*, the next procedure is to practise this sound in association with various vowels, the latter respectively preceeding and succeeding the consonant. Finally comes the double consonant, the *sk*, *sl*, *sm*, etc., and these sometimes offer at first some considerable difficulty, but this can be overcome by taking each consonant separately and then conjoined.

In only one of my cases can I claim that perfect speech *in all circumstances* has been secured as the combined result of operation and speech training, and this patient belonged to *Group* 3 in which no consonants and no fault beyond the glottic stop had been acquired; before operation he was quite unintelligible.

Although speech training will improve the patient beyond all recognition, it will never produce perfect speech, since the acquisition of this depends entirely on the development of a habit and represents a tremendous uphill fight.

Group 3 speakers possess only the vowels, which are used with or without the glottic stop. This group includes a curious mixture of cases in which speech sense and mental dullness are encountered in varying proportions. Mention has been made previously of a patient in this group who within three months of operation was sound perfect and ultimately developed into an habitual normal speaker (*Case* 22). Unfortunately not all in this group display such speech sense, as it is quite a common experience to meet with the type who is quite unable to imitate even the simplest sounds, and it is very difficult to be sure whether this is the result of a failed operation or just stupidity. One thing is quite certain—namely, that the type of cleft present has no real influence on which particular group of speech the patient will adopt.

PHARYNGOPLASTY AS A SECONDARY OPERATION.

There is an enormous number of cases in this country in which the palate has been repaired by one method or another, but in which no functional valve has been formed. Every speech-training clinic is filled with such patients, and much valuable time and energy is wasted in attempting to restore normal speech to those who do not possess the necessary mechanism—an effort which can only be compared to attempting to obtain music from a broken flute. Yet it is possible in some of these cases to restore the palato-pharyngeal valve and thus ensure that at any rate the flute is repaired in an efficient manner, with consequent improvement in speech and the chance that the patient may, should he or she so desire, acquire perfect speech. Unfortunately the opportunities of carrying out work of this kind are few and far between, since patients do not readily submit to further operation when they have tasted failure from a previous operation done for the same condition.

However, I have had the opportunity of performing pharyngoplasty on ten occasions where the palate had previously been successfully repaired by others and the speech result was bad. Although a considerable degree of thought has been devoted to this aspect of cleft-palate surgery, the amount of experience afforded has not allowed of any firm conclusions with regard to technique in the treatment of the palate itself. One fact stands out quite clearly, however—namely, that pharyngoplasty is capable of restoring a valvular mechanism, and in the right type of case causes immense improvement in speech. Of the 10 cases mentioned, only 2 possessed any mobility in the soft palate, all the remainder being firmly fixed as a result of previous interference. Of these 10 cases, 3 live in distant parts of the country and have consequently not been submitted to personal observation since operation, and 1 lives nearer but has not presented himself for review. Of the remaining 6, 4 present evidence of palatopharyngeal valves and 2 are complete failures.

Pharyngoplasty after repair of the palate presents no serious difficulties, as the soft palate can easily be held forward by means of a small hook or an aneurysm needle and the operation can be completed as in the unrepaired case. All attempts to secure mobility in the soft palate have in my hands failed, although I believe that by division of the hamular processes and freeing the posterior end of the hard palate a little more has been secured in length. Further length in the palate has also been obtained by paring the free edges of the posterior pillars of the fauces for about $\frac{1}{3}$ in. and then suturing in the mid-line. This brings the posterior pillars into great prominence, and the uvula projects forwards in a nipple-like fashion, while any remaining tonsillar tissue points in a more forward direction.

The 6 cases in which secondary operations have been done all presented themselves with immobile soft palates, so that the problem of restoring the palatopharyngeal valve was materially increased. These cases are worth studying in detail.

Case 25 was that of a girl aged 9 years. She had had a wide median cleft of the soft and hard palates almost reaching the alveolar margin. Several operations of the classical type done previously had resulted in

complete fixation of the soft palate and there was a large hole in the hard. Speech was of the type noted as *Group* 2—that is, with the development of all the faults. Pharyngoplasty was done in June, 1931, and as a result speech was much improved. At a second operation the mucoperiosteum of the hard palate was stripped from the bone in the form of two flaps based on the posterior palatine artery, and the hamular processes were destroyed, the operation being completed by suture in the middle line. The improvement in speech was immediate. This child can now snort strongly; her speech is still of the cleft-palate type, but in my presence she was able to make the principal consonants without any nasal escape whatever. The *s* was of the cleft-palate type and could be made with the mouth wide open, but after a few minutes' demonstration she was able to make it the correct way and without nasal escape. This child has been undergoing training, but not under my supervision.

Case 32 was a girl aged 14 years who had a median cleft of the soft and hard palates repaired at the ages of 2 and 2½ years. After the first operation the palate broke down completely, but held after the second attempt. The soft palate was completely immobile, but the speech was very good, the only defective sounds, apart from the gross nasal escape, being the *k* and *g*. At operation in May, 1931, pharyngoplasty was done by the usual method, and it was noted that immediately afterwards speech improved immensely; but unfortunately this was not completely maintained owing to some of the sutures giving way. At a second operation in September, 1932, a further attempt was made to increase the pharyngeal narrowing of the first operation, and the posterior edges of the soft palate and posterior faucial pillars were pared and sutured in the mid-line. Unfortunately all the sutures gave way and there was no further improvement. As a result of all this operating the patient is able to snort, but only weakly; her speech is very much better, but she is still unable to effect fully competent pharyngeal closure.

Case 39 was a boy aged 7 years. He had had a bilateral complete cleft of the hard palate with a double hare-lip operated on within three years of birth. When I first saw him his speech was quite unintelligible. The palate was rigid and lay far from the pharyngeal wall. At operation in 1931 the mucoperiosteum of the hard palate was stripped up in the form of two flaps based on the posterior palatine artery and the hamular processes were destroyed. A large hole was left at the anterior end of the hard palate and this was later closed by an obturator. A very satisfactory pharyngoplasty was done at the same time. There was some immediate improvement in speech, but as it was not felt that the maximum possible surgical closure had been effected, a second operation was decided upon and carried out early in 1932, when the pharyngoplasty was further tightened and the uvula and the posterior edges of the soft palate and posterior pillars of the fauces were pared and sutured in the mid-line. This boy has been under constant observation since and has been trained by Mr. T. W. Moles. Although he was an unintelligible *Group* 2 speaker at first, he is now able to obtain every sound perfectly. He can snort very strongly and can inflate a rubber balloon. Training has been discontinued. His father writes, "Mr. Moles is of the opinion that I can now complete the training myself as G—— can get all

the sounds *when he likes* and it is now mainly a matter of continued exercise." The italics are the father's.

Case 72 was aged 5 years at the time of operation. He had a complete unilateral cleft of the palate with a hare-lip which had been operated upon within two years of birth. On first being seen, the cleft was completely closed but the soft palate was immobile. At operation in June, 1928, pharyngoplasty was done. The boy was not seen for over four years, when it was found that he had a perfectly competent valve which enabled him to inflate a balloon in my presence. Speech was still of the cleft-palate type in *Group* 2, but no training of any sort had been given, yet in my presence he could be made to say many consonants correctly.

Case 33 was aged 12 years. He had had a median cleft of the soft and hard palates which had been repaired, resulting in a hopelessly immobile soft palate with much scarring. The nasopharynx was of enormous proportions. Speech was in *Group* 3 and quite unintelligible. Pharyngoplasty was carried out but made no improvement, the boy being unable to imitate any sound whatever. It should have been recognized before operation that he was hopelessly dull mentally and not worth any waste of time or effort.

Case 46 was a woman aged 24 years with a previously repaired bilateral cleft. The lip was extremely bad and an enormous hole existed in the hard palate. The soft palate was immobile and short. Speech consisted of vowel sounds with the glottic stop and an occasional consonant. Pharyngoplasty was done in April, 1932, and although it appeared to be a very satisfactory surgical result there is no evidence of valve formation nor is speech improved.

Comment.—In the cases outlined above 4 out of the 6 have now palato-pharyngeal valves, although in *Case* 32 the valve is not fully competent, a fact which is supported by the weakness of the snort. Three of the cases present no doubt as to the efficiency of the valve, and this in spite of an immobile palate, and it is interesting to observe how the ridge of a pharyngoplasty is able to compensate, at any rate in the simpler exercises, for this loss. The opportunity of operating upon and following the subsequent progress of a case with a freely mobile palate is awaited with interest.

REFERENCES.

[1] *Jour. of Anat.*, 1930, lxv, Pt. 1, October.
[2] *Brit. Jour. Surg.*, 1928, xvi, 127.
[3] PAGET, SIR RICHARD, Bart., *Human Speech*, 1930. London: Kegan Paul, Trench, Trubner, & Co. Ltd.

SHORT NOTES OF RARE OR OBSCURE CASES

GRANULOMA OF INTESTINE. STENOSIS OF ILEOCÆCAL VALVE.

By H. W. L. MOLESWORTH, Folkestone.

A woman aged 30 had had an appendicectomy done in 1921 by another surgeon. At that time she complained of vomiting and slight right-sided abdominal pain; these symptoms were relieved by the operation, and she remained perfectly well until July 4, 1932, when she had a short attack of colicky abdominal pain associated with nausea, but without vomiting or constipation. This lasted for a few days and she got completely well.

Towards the end of July she underwent an operation for hallux valgus; she was in hospital in London and during this time complained of no abdominal symptoms. On Aug. 18 she complained of colicky abdominal pain and nausea. Up to Aug. 23 the bowels acted with regularity. On Aug. 24 pain was much more severe. The patient was seen by me that evening and was then in a condition of obstruction to the small intestine.

A laparotomy was done the same evening. The small gut was distended and hypertrophied and there was a swelling in the region of the ileocæcal valve which somewhat resembled that found in congenital pyloric stenosis. The terminal few inches of the mesentery of the intestine were markedly thickened and indurated, as was the terminal portion of the small gut. An anastomosis was made between the small intestine and the transverse colon. At this stage there were no adhesions connected with the small intestine. Those that exist on the specimen subsequently removed have occurred since the first operation.

On Sept. 9 the abdomen was re-opened, and removal of the ileum, ascending colon, and about one-third of the transverse colon carried out. The operation was rendered difficult by numerous adhesions between the ascending colon and the small intestine. The patient has made a good recovery and is well at the time of writing.

The drawings by Mr. Sewell (*Figs.* 259, 260) show the condition found, and Mr. Lawrence has kindly permitted me to make use of his description of the specimen, which is in the Museum of the Royal College of Surgeons:—

"*Stenosis of ileocæcal valve:* The terminal 6 in. of the ileum, with the cæcum, ascending colon, and first part of the transverse colon, which were removed by operation. An incision has been made in the free border of the ileum and cæcum, displaying a tight stricture of the ileocæcal valve, admitting only of a narrow probe. The stricture is about $\frac{3}{4}$ in. in length and the wall of the intestine bounding it is thickened to the extent of $\frac{1}{2}$ in., the thickening

FIGS. 259, 260.—Specimen removed at operation, with the ileum and cæcum shown open below. A, Dilated and hypertrophied ileum; B, Polypous mucosa; C, Organizing granulation tissue; D, Stricture; E, Cæcum; F, Ascending colon; G, Transverse colon.

being due mainly to a fibrous-looking increase of the submucous layer; the thickening is continued for 2 in. along the adjoining part of the ileum. The lining of the stenosed segment is the seat of small polypoid excrescences. Partly covering the stenosed portion on the external surface of the bowel is a mass of dense fibro-fatty tissue measuring 1½ in. in width. The vermiform appendix is absent. The terminal part of the ileum is dilated and hypertrophied; its lining is irregularly thickened, and exhibits small papillary projections in places. Its surface is covered with dense adhesions, a delicate membranous adhesion also covers the cæcum and ascending colon; the mucous membrane of the latter is free from ulceration. No enlarged glands are present in the specimen.

"Microscopical examination (*Fig.* 261): The mucous membrane is ulcerated on both sides of the stenosis. On the surface there is a layer of coagulated pus, beneath which is a broad zone of granulation tissue.

FIG. 261.—Microscopical section of the wall of the ileum. (× 25.)

A thick formation of fibrous tissue replaces the submucous layer, and the two muscular coats are hypertrophied and separated by a similar formation. Both fibrous and muscular layers contain numerous foci of leucocytes, mostly arranged around vessels. At the margin of the ulcer the mucous membrane is raised in the form of small polypi or papillomata; these are covered with cylindrical epithelium, among which are numerous goblet cells. There is no evidence of carcinoma; no downgrowth of epithelium at the base or margin of the ulcer.

"It is probable that the stenosis of the ileocæcal valve was a sequela of the appendicitis and appendicectomy."

This condition would appear to be identical with that described by Mock,[1] and more recently by Erdmann and Burt,[2] whose articles contain a list of references to the literature.

REFERENCES.

[1] MOCK, *Surg. Gynecol. and Obst.*, 1931, lii, 672.
[2] ERDMANN and BURT, *Ibid.*, 1933, lvii, 71.

A SOLITARY PLASMOCYTOMA OF THE FEMUR.

By CYRIL POLSON and N. SHIRES,

FROM THE DEPARTMENTS OF PATHOLOGY AND BACTERIOLOGY OF THE UNIVERSITY AND ST. JAMES'S HOSPITAL, LEEDS.

During 1932 two publications, those of Stewart and Taylor,[1] and Harding and Kimball,[2] concerned solitary plasmocytoma of bone. Each reviewed the literature, giving indication of the rarity of this neoplasm. The former authors described two original cases, one a plasmocytoma of the humerus and one which probably arose in the maxilla, while the latter authors described a plasmocytoma of the femur.

In the case described by Rogers,[3] the patient, who had a solitary plasmocytoma of the right femur, came under observation on account of pathological fracture of the bone following a slight accident. Amputation of the leg resulted in his complete recovery.

The present case resembles that of Harding and Kimball, since it was a growth of the femur at first considered a secondary deposit of carcinoma.

History.—The patient was a male aged 72 years who was admitted to St. James's Hospital on Sept. 5, 1928, on account of chronic gastritis and hæmorrhoids. He was re-admitted on Oct. 4, 1932, because he had slipped on the stairs at his home and fractured his left femur. During the previous fortnight he had had pain in this leg and used a stick when walking. He also complained of irregularity of the bowels and difficulty in micturition.

Fig. 262.—Radiogram of left femur showing evidence of rarefaction but no expansion of the bone in the affected area.

On Admission.—The patient had a fracture in the middle third of the left femur, and after radiological examination Dr. Wall reported that this was a pathological fracture (*Fig.* 262). The leg was placed on a splint. No examination of the urine for the detection of Bence-Jones protein was made. Twelve days after admission (Oct. 16) his abdomen became severely distended, but no tenderness or rigidity was apparent. He vomited, and an enema produced no result. A diagnosis of intestinal obstruction, probably due to a carcinoma of the bowel, was made.

Operation.—The abdomen was opened under local anæsthesia. The large intestine was grossly distended and a transverse colotomy was performed.

The circumstances precluded any extensive examination of the peritoneal cavity. Although some improvement followed, the patient died on Oct. 20.

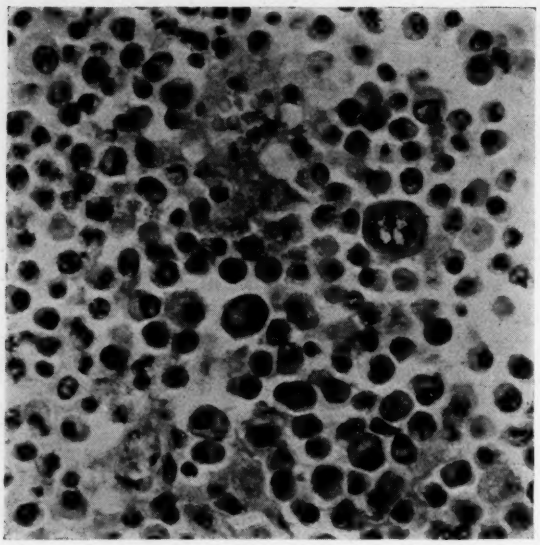

FIG. 263.—Microphotograph showing types of cell. A large 'pre-plasmocyte' can be seen in the right upper quadrant. (× 100.)

POST-MORTEM FINDINGS.—At autopsy the body was that of a well-nourished elderly man. His left leg was shorter by 3 in. than the right and externally rotated. No external abnormality of the left thigh was observed nor was any tumour palpated, but upon manipulation abnormal mobility was present at the middle of the femur. Examination of the bone showed that there was an ununited fracture in its middle third with over-riding of the fragments. In the process of clearing the bone of its muscle sheath, yellowish growth, infiltrated with blood, was encountered on the outer aspect of the bone and adjacent to it in the region of the fracture. The mass of growth was small and was less than 3 in. in diameter. Little or no expansion of the bone had occurred, but there was destruction of about 1 in. of the shaft in the region of the fracture. Rarefaction of the bone was present for a distance of about 2 in. above and 1 in. below the fracture, and here the medullary cavity was filled with growth of a soft consistency and greyish colour. Death was due to acute perforation of a chronic duodenal ulcer situated on the anterior wall. There was but little peritonitis or escape of fluid. Except for one or two small adenomata of the prostate no other neoplasm was found. Early bronchopneumonia was present as a terminal condition.

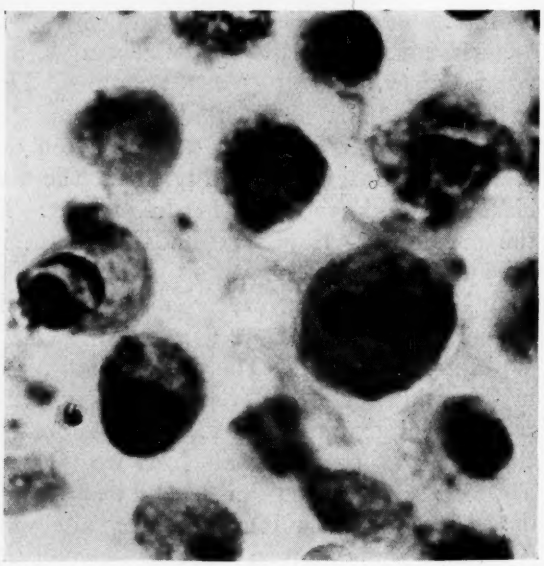

FIG. 264.—Microphotograph showing plasma cells, one containing a mitotic figure. (× 1200.)

MICROSCOPICAL FINDINGS.—The femoral tumour was subjected to histological examination and proved a plasmocytoma. Two types of cell were abundant. First an ovoid or pyriform cell having an eccentric nucleus containing plentiful

chromatin frequently arranged in 'cart-wheel' pattern (*Fig.* 263). The protoplasm yielded the characteristic reaction with Pappenheim's stain, and a perinuclear halo was observed in a small proportion of these cells. They were plasma cells, but of rather larger size than those usually seen in chronic inflammatory foci. The second type of cell was even larger and possessed a large nucleus, centrally situated (*Fig.* 263). The protoplasm yielded the Pappenheim reaction, and these cells are considered to be immature plasma cells.

Mitosis was frequent in certain areas of the growth (*Fig.* 264). A count of approximately 7500 cells in 100 adjacent fields viewed with the $\frac{1}{12}$ objective showed that mitosis occurred in 1 per 200 cells. Several phases of mitosis were observed, and the cells concerned yielded the Pappenheim reaction, although their protoplasm was a little paler than that of other plasma cells. Multinucleated cells and lymphocytes were scarce and distributed through the growth. There was a sparse collagenous stroma in which were a number of blood-vessels, most being well formed. As in Stewart and Taylor's cases, the stroma fibrils were, in some areas, arranged in parallel with columns of plasma cells between. The areas of hæmorrhage were ascribed to the violence of fracture.

COMMENTARY.

It is scarcely a matter for surprise that the diagnosis of the femoral lesion was not made during life. Moreover, even in the presence of much less evidence calculated to mislead, it is not unlikely that a plasmocytoma may be mistaken for a secondary deposit of carcinoma. It is imperative that these two conditions should be differentiated, since the local malignancy of solitary plasmocytoma has been demonstrated and since local treatment may result in a complete cure. The necessity of a biopsy before proceeding to radical surgery has been insisted upon by Stewart and Taylor, and the present case bears out this conclusion in an unmistakable fashion. Harding and Kimball comment upon the fact that plasmocytoma of the appendicular skeleton invariably causes pathological fracture. We therefore insist that the reasonable procedure in all cases of pathological fracture is an open examination of the lesion and the removal of tissue for biopsy. Should it be a secondary deposit of carcinoma, then the prognosis is determined, whereas if it is a solitary plasmocytoma, much may be done, not only to save the patient's life, but also to restore him or her to health and activity.

Since a complete examination of the skeleton was not made in this case, the tumour was not proved to be a solitary plasmocytoma, although this was almost certain. The essential histological features of the growth were in accord with those described by other authors, but there was evidence of unusual activity, since large 'pre-plasmocytes' were plentiful and numerous mitotic figures were seen.

We are indebted to Dr. J. Wall for permission to reproduce the radiogram.

REFERENCES.

[1] Stewart, M. J., and Taylor, A. L., *Jour. Pathol. and Bacteriol.*, 1932, xxxv, 541.
[2] Harding, W. G., and Kimball, T. S., *Amer. Jour. of Cancer*, 1932, xvi, 1184.
[3] Rogers, H., *Brit. Jour. Surg.*, 1930, xvii, 518.

REVIEWS AND NOTICES OF BOOKS.

Poliomyelitis. A Survey made possible by a Grant from the International Committee for the Study of Infantile Paralysis organized by Jeremiah Milbank. Large 8vo. Pp. 562 + xxii, with 25 plates. 1932. Baltimore: The Williams & Wilkins Company. (London: Baillière, Tindall & Cox.) 35s.

THIS volume represents four years' work, carried out by grants from the Fund, of forty-four practical workers at a cost of 280,000 dollars. It contains a comprehensive survey of poliomyelitis so far as the history, etiology, symptoms, pathology, and epidemiology are concerned. But it omits treatment entirely except prophylaxis and the treatment of the acute attack by serum and other similar measures. The references are very extensive, occupying 43 pages, and as there is no indication to show which portions of the work summarized have been carried out by the investigators who have received grants from the Committee, it is difficult to form any opinion as to whether the results are really commensurate with the amount of work done and the cost incurred.

The chapter on epidemiology is very full and of much interest. It is noticeable that, whereas in Sweden the case rate per 100,000 has risen above 10 on several occasions, with a peak of 70, and that the rates have also been high in the United States and New Zealand, in England this rate has never been as high as 5. In the opinion of the Committee there has been no proof that the administration of convalescent serum in any way alters the severity of the attack or the prognosis as to the severity of the resulting paralysis. This does not appear to agree with observations in other countries.

The book will be consulted by those who are interested in research work upon this disease, but is of no real value to a clinician.

The Principles of Treatment of Muscles and Joints by Graduated Muscular Contractions. By MORTON SMART, C.V.O., D.S.O., M.D., Ch.B. (Edin.), Late Medical Officer in Charge of the Electrotherapeutic Department, Hospital for Sick Children, Great Ormond Street. Large 8vo. Pp. 217 + xvi. 1933. London: Humphrey Milford. 15s. net.

MORTON SMART'S method of treatment by graduated muscular contractions, produced by stimulation with a faradic coil of special pattern, is now very widely known and practised. This personal and authoritative description of his methods is therefore very valuable, and it is essential that it should be read by all those who practise physical treatment. In the earlier chapters there is a general account of the physiology of muscular action and the pathology of some of the conditions treated. Following this, manipulation of a shoulder-joint for adhesions and after-treatment are described and then a general account of muscle treatment is given. No doubt there will be many readers who do not agree entirely either with the author's methods or with his opinions. For example, to force abduction and rotation of the shoulder directly (without traction) may be a dangerous form of manipulation, risking fracture if there are firm adhesions. Morton Smart believes that faradic stimulation of muscles will increase their strength and volume; many will hold that voluntary action will do so more successfully. And many will feel that to give as large a number as 1800 contractions to a muscle at each sitting may well mean overworking it if it has been disused. In general also we may believe that the faradic treatment of muscles should be largely a preparation for the use of active movements and exercises, a side of the work which is not stressed in this volume.

However, these criticisms do not detract from the value of the book in its description of methods and in the final chapter of details of the apparatus used.

Treatment of Fractures in General Practice. By W. H. OGILVIE, M.D., M.Ch., F.R.C.S. F'cap 8vo. Vol. I, Pp. 108 + viii; Vol. II, 109–180. Illustrated. 1932. London: John Bale, Sons & Danielsson Ltd. 2s. 6d. each.

THIS little book is an excellent example of how much can be compressed into a small space. It contains an outline of the anatomy, including development of epiphysial nuclei, the mode of causation, pathology of repair, and treatment of fractures, all put in a form which can very easily consulted by a general practitioner.

Naturally in the case of fracture treatment the methods are those of the author and would not necessarily be accepted in detail by everyone, but there is little to be found fault with. The principles are sound throughout and methods described with sufficient detail and with simple but adequate illustrations. Mr. Ogilvie has introduced the name of "cancellous fracture" in injuries of such bones as the os calcis and vertebral bodies instead of the term "crush fracture" which is more often applied. One omission is that of any description of the use of the X-ray operating table in the setting of fractures. The illustration of a Colles's fracture after reduction shows the wrist in an extended position, a method which would be generally condemned, and under the treatment of delayed union the use of parathormone is advised. Considering that the latter is now known to cause decalcification of the bones its use for this purpose should not be advocated in a modern text-book.

The Injured Workman. By G. F. WALKER, M.D., M.R.C.P., with the collaboration of J. HARVEY ROBSON, Barrister-at-Law (Law); R. E. JOWETT, M.D., Ch.B., D.L.O. (Ear, Nose, and Throat Surgery); STANLEY RITSON, M.B., M.S., B.Sc., F.R.C.S. (General Surgery); JOHN FOSTER, M.A., M.B., F.R.C.S., D.O.M.S. (Ophthalmic Surgery). Foreword by W. H. MAXWELL TELLING, M.D., B.S., F.R.C.P., Professor of Forensic Medicine, University of Leeds. Crown 8vo. Pp. 190 + xx. 1933. Bristol: John Wright & Sons Ltd. 6s. net.

THIS work deals with the legal aspects of the late results of an accident to a workman, and by 'accident' is here meant a single blow, stress, or injury. An impressive foreword is contributed by Dr. W. H. Maxwell Telling, who rightly points out the great difficulty that exists in finding 'light work' for the injured workman during the stage of transition from partial to complete recovery—a period when the most important factor in therapeusis is to get him back to 'something' in the way of work as soon as possible.

In the first chapter the essential features of the Workmen's Compensation Acts are well epitomized, but in other parts of the volume it is not sufficiently emphasized that compensation is awarded not because an injury has been received as the result of an accident arising out of and in the course of employment, but because that injury has resulted in an actual loss of earning capacity. The section on "Rusting and Brooding" in Chapter II may be especially commended for its philosophic thought and its sound common sense.

The special injuries of the various regions of the body are considered in detail, but here a sense of proportion seems to have been wanting. Thus, of a total of 128 pages devoted to the whole of the body no fewer than 50 are concerned with the eye—injuries of which form a relatively uncommon cause for compensation—while the infinitely more important injuries to the fingers and hands are very summarily dismissed.

The Cure of Hæmorrhoids, Varicose Veins and Ulceration, and Allied Conditions: by Modern Methods and Bandaging. By STUART MCAUSLAND, B.A. (Lond.), M.D., Ch.B. (Liv.), M.R.C.S., L.R.C.P., Hon. Physician, Hahnemann Hospital, Liverpool, etc. Large 8vo. Pp. 63. Illustrated. 1933. London: John Bale, Sons and Danielsson. 3s. 6d. net.

THIS is a small handbook on the injection treatment of varices and hæmorrhoids. It is well illustrated and gives some practical suggestions as to the methods, drugs, and instruments used. It will serve as a cheap and useful guide to the general practitioner.

The Diagnosis and Treatment of Postural Defects. By WINTHROP MORGAN PHELPS, B.S., M.D., M.A., F.A.C.S., Professor of Orthopædic Surgery, Yale University, etc.; and ROBERT J. H. KIPHUTH, Assistant Professor of Physical Education, Yale University. Royal 8vo. Pp. 180 + xiv, with 108 illustrations. 1933. London: Baillière, Tindall & Cox. 23s. net.

AN account of the personal views of the authors upon posture and upon corrective exercises. The latter are not based upon the Ling system, and for this reason alone the book is not likely to be read much in England. Moreover, many of the views on posture would appear to be based on pure mechanics rather than on physiological principles. 'Stretch and strengthen' rather than re-education are emphasized. Many people fail to realize the value of Ling's methods because thay have not studied them.

Diseases of the Spinal Cord. By WILLIAMS B. CADWALADER, M.D., Professor of Clinical Neurology, University of Pennsylvania Medical School, etc. Royal 8vo. Pp. 204 + xviii, with 72 illustrations. 1932. London: Baillière, Tindall & Cox. 29s.

THIS book gives an adequate and reasonably compact account of the diseases of the spinal cord. It has no definite surgical orientation and therefore does not demand detailed review here. To those who are called upon to apply surgical methods to cord affections it can be recommended as giving a satisfactory picture of spinal neurology. There is a bibliography of 328 items.

The Digestive Tract. A Radiological Study of its Anatomy, Physiology, and Pathology. By ALFRED E. BARCLAY, O.B.E., M.A., M.D., D.M.R.E., Lecturer in Medical Radiology, University of Cambridge. Large 8vo. Pp. 395 + xxviii, with 275 illustrations. 1933. Cambridge: University Press. 36s. net.

THIS volume is the most complete account of the radiographic appearances of the gastro-intestinal tract that has yet been published in this country. While the book is of special interest to radiologists, for whom the technical side of the matter appears to have been very thoroughly gone into, there are separate sections on the anatomy and physiology of the tract, in which the author elaborates his theory of the fluidity of the abdominal contents, and in which he shows that the position of the stomach varies widely under the influence of various stimuli, even during the course of an examination. Some of our accepted views on the anatomy and physiology of the stomach are bound to be modified by a perusal of this volume, and it may be that the debt which the surgeon owes to radiology will have to be shared in future by his colleagues in the anatomical and physiological schools.

The book is clearly printed, and the numerous radiograms are finely reproduced. The style is fluent and easy, and the author seldom fails to express himself clearly. The book, as a whole, must be regarded as the finest contribution so far made by radiology in this country to our knowledge of the gastro-intestinal tract.

Atlante di Chirurgia dello Stomaco. By G. EGIDI. Large 8vo. Pp. 15, with 30 plates. 1933. Rome: Luigi Pozzi. L. 35.

THIS little atlas of the surgery of the stomach is a credit to the Roman publishing house of Luigi Pozzi. Lying upon the table, it might well be a portfolio of etchings, and indeed the drawings are those of an artist.

A few anatomical plates suffice to make clear the points of entry for injection of anæsthetic fluids, if that method be preferred, and the vascular supply that must be controlled or respected by the operator. One plate displays the sutural methods adapted to gastro-intestinal junctions, and the rest illustrate gastrostomy, gastrotomy, Finney's and Rammstedt's operations, gastro-enterostomy, several types of partial gastrectomy, total gastrectomy, and interventions for gastrojejunal ulcer and gastrocolic fistula. A separate running commentary elucidates the points of the pictures and records the author's predilections as to details.

Roentgenographic Studies of the Urinary System. By WILLIAM E. LOWER, M.D., F.A.C.S., Chief of Department of Urology, Cleveland Clinic, etc.; and BERNARD H. NICHOLS, M.D., F.A.C.R., Chief of Department of Roentgenology, Cleveland Clinic. Imperial 8vo. Pp. 812 + xviii, with 812 illustrations. 1933. London: Henry Kimpton. 84s. net.

THE rapid advance of urology during the last few years and in particular the development of intravenous pyelography should ensure a welcome for this book. Until recently pyelography could only be carried out with the assistance of surgeons who had the necessary experience in cystoscopy, but the introduction of uroselectan has brought it within the reach of all who can inject the solution into a vein and take skiagrams. The radiology of the urinary tract, however, is not a simple matter; not only are there many essentials in the technique which must be mastered before good and reliable results can be obtained, but also the interpretation of these results is often difficult and calls for considerable experience. Unless the technique is good and the interpretation correct, serious mistakes may be made.

The first part of this book is devoted to an excellent account of the technique of the radiological examination of the various parts of the urinary tract, with a brief description of the pathological conditions and abnormalities which may be found. The second part contains some 750 skiagrams of 443 cases, accompanied by a summary of the history and clinical findings, together with an interpretation of the skiagrams in each case. Most of the skiagrams are admirably clear, and their value is increased by a pen-and-ink sketch illustrating the main points of interest. Some of the intravenous pyelograms, however, are ill defined and might with advantage be replaced by others in the next edition.

There are a few errata—'corpus cavernosa' on page 11, 'hypotonic' for 'hypertonic' on page 55, 'epiploicea' on page 61, 'blood' for 'bladder' on page 792, and 'left kidney' for 'right kidney' on page 800B.

This book, written as it is by a urologist and radiologist in collaboration, should prove of great value to those whose experience in urography is not extensive, and will be read with profit by all who are interested in urinary surgery.

BOOK NOTICES.

[*The Editorial Committee acknowledge with thanks the receipt of the following volumes. A selection will be made from these for review, precedence being given to new books and to those having the greatest interest for our readers.*]

The Thyroid Gland: its Chemistry and Physiology. By CHARLES ROBERT HARRINGTON, M.A., PhD., F.R.S., Professor of Pathological Chemistry in the University of London. Large 8vo. Pp. 222 + xiv. Illustrated. London: Oxford University Press. 15s. net.

Intracranial Tumours. By PERCIVAL BAILEY, Professor of Surgery, University of Chicago. Royal 8vo. Pp. 475 + xxiv, with 155 illustrations and coloured plates. 1933. London: Ballière, Tindall & Cox. 35s. net.

Paralysis in Children. By R. G. GORDON, M.D., D.Sc., F.R.C.P. (Ed.), Physician, Bath and Wessex Orthopædic Hospital, etc.; and M. FORRESTER-BROWN, M.D., M.S. (Lond.), Surgeon, Bath and Wessex Orthopædic Hospital, etc. Demy 8vo. Pp. 328 + viii, with 116 illustrations. 1933. London: Humphrey Milford. 15s. net.

Surgery of the Stomach and Duodenum. By J. SHELTON HORSLEY, M.D., F.A.C.S., LL.D., Attending Surgeon, St. Elizabeth's Hospital, Richmond, Va. Royal 8vo. Pp. 260, with 136 illustrations. 1933. London: Henry Kimpton. 35s. net.

Actinotherapy Technique. An outline of indications and methods for the use of modern light therapy. With a Foreword by Sir HENRY GAUVAIN, M.D., M.Chir. (Camb.), F.R.C.S. Crown 8vo. Pp. 184. Slough: The Sollux Publishing Co. 6s. net.

St. George's, 1733-1933. By J. BLOMFIELD, O.B.E., M.D. F'cap 4to. Pp. 120, with 14 plates. 1933. Published for St. George's Hospital by the Medici Society, London. 5s. net.

The Enlarged Prostate and Prostatic Obstruction. By KENNETH M. WALKER, F.R.C.S., M.A., M.B., B.C., Lecturer in Venereal Diseases, St. Bartholomew's Hospital, etc. Second edition. Demy 8vo. Pp. 223 + xiv, with 63 illustrations. 1933. London: Humphrey Milford (Oxford University Press). 14s. net.

History of Urology. Prepared under the auspices of the American Urological Association. Editorial Committee: E. G. BALLENGER, W. A. FRONTZ, H. G. HAMER, B. LEWIS. Two volumes. Royal 8vo. Vol. I. Pp. 386 + xii, with 46 illustrations. Vol. II. Pp. 362 + vii, with 12 illustrations. 1933. London: Baillière, Tindall & Cox. 36s. net.

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